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Archives of Neurology and Psychiatry

VOLUME 18 JULY, 1927 NUMBER 1

DECEREBRATE RIGIDITY FOLLOWING ENCEPHALITIS

REPORT OF A CASE WITH NECROPSY *

THEODORE H. WEISENBURG, M.D.
AND
BERNARD J. ALPERS, M.D.
PHILADELPHIA

Postencephalitic muscular rigidity is not an uncommon occurrence and is seen most frequently in the parkinsonian states. Here it manifests itself as a mild to severe form of hypertonia with poverty of movement, relative motor weakness, parkinsonian attitude and other characteristics pointed out recently by Wilson.[1] Occasionally, however, the rigidity that occurs after epidemic encephalitis is of such extreme degree as to make it the outstanding feature; it is as an illustration of this that our case is reported, the rigidity closely simulating decerebrate rigidity. In addition, the absence of pathologic reports in these cases makes such records desirable.

That marked rigidity may occur as a sequel of other organic diseases than encephalitis has already been demonstrated both clinically and pathologically. Antheaume and Trepsat,[2] in 1920, reported a case of general paralysis in a patient, aged 43, with catatonia, cataleptic states and stereotypy. In 1923, Urechia and Elekes [3] reported four cases of general paralysis with generalized rigidity and presumed the lesion to be in the globus pallidus; more recently, Steck [4] reported the occurrence

* From the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases, the Laboratory of Neuropathology, Philadelphia General Hospital, and the Graduate School of Medicine, University of Pennsylvania.

* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June 1, 2 and 3, 1926.

1. Wilson, S. A. K.: Disorders of Motility and Muscle Tone, Lancet **2**:1, 1925.

2. Antheaume, A., and Trepsat, L.: General Paresis and Catatonia, Encéphale **15**:297, 1920.

3. Urechia, C. I., and Elekes, N.: La rigidité pallidale syphilitique, Encéphale **18**:503, 1923.

4. Steck, H.: The Striatal Symptom-Complex in General Paresis, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:424 (July) 1925.

of catatonia in fourteen of nineteen patients with general paralysis, the lesion being in the corpus striatum. In addition catatonia and catatonic states have been described in uremia and in other conditions.

CASES OF MARKED RIGIDITY IN ENCEPHALITIS

Most of the cases of marked rigidity in encephalitis up to the present time have been described as cases of catatonia or catalepsy. Hunt [5] briefly describes a cataleptic type of encephalitis, and remarks that "the association of general muscular rigidity of the paralysis agitans type with the peculiar lethargy characteristic of epidemic encephalitis produces a clinical picture closely resembling catalepsy (flexibilitas cerea)." Wilson [6] gives a clinical report of a case of the catatonic type of epidemic encephalitis in a patient, aged 31, who often maintained a curious fixity of attitude for indefinite periods. Bassoe [7] states that in severe cases of encephalitis "the whole body becomes rigid and all movements slow and labored, the condition resembling both catatonia and advanced parkinsonian rigidity." He attributes all the symptoms to involvement of the extrapyramidal system. Sicard and Bollack [8] report a case of catatonia in a postencephalitic case resembling parkinsonism but with more marked rigidity than is usually seen in this condition. Tilney and Howe [9] describe two cases with unusual hypertonus in the muscular system determining fixed positions of the trunk and limbs, and producing a rigidity that had some flexibilitas cerea. There was no involvement of the corticospinal system. Barker [10] has briefly mentioned the occurrence of cataleptic or catatonic states in epidemic encephalitis and reports, in brief, the case of a patient who made a good recovery. Wimmer [11] in his recent monograph merely mentions that catatonic attitudes may occur in encephalitis and reports a case. Mourgue [12] cites decerebrate rigidity in a case of torsion spasm follow-

5. Hunt, Ramsay: Epidemic Encephalitis. Report of Association for Research in Nervous and Mental Disease, New York, Paul Hoeber, 1918.

6. Wilson, S. A. K.: Epidemic Encephalitis, Lancet **2**:7 (July 6) 1918.

7. Bassoe, P.: Epidemic Encephalitis (Nona), J. A. M. A. **72**:971 (April 5) 1919.

8. Sicard, M., and Bollack, M.: Catatonies, hypertonies, attitudes fugues au cours de l'encéphalite léthargique, Bull. et mém. Soc. méd. d. hôp. de Paris **44**:262, 1920.

9. Tilney, F., and Howe, H. M.: Epidemic Encephalitis, New York, Paul Hoeber, 1920.

10. Barker, L. F.: Diagnostic Criteria in Epidemic Encephalitis and Encephalomyelitis, Arch. Neurol. & Psychiat. **6**:173 (Aug.) 1921.

11. Wimmer, A.: Chronic Epidemic Encephalitis, London, William Heinemann, 1924.

12. Mourgue, R.: Le syndrome clinique de la rigidité décérebrée de S. A. K. Wilson dans un cas de spasme de torsion consécutif à l'encéphalite epidémique, Schweiz. Arch. f. Neurol. u. Psychiat. **11**:163, 1922.

ing encephalitis. His patient, a man aged 35, had had an attack of encephalitis, and on walking developed rigidity and hyperextension of the arms, opisthotonos, hypertonicity of the muscles of the neck, and extension of the legs so that he walked on his toes. In addition, there were torsion movements of the left arm on its axis, torsion movements of the neck to the left, lordosis and scoliosis. Walking on all

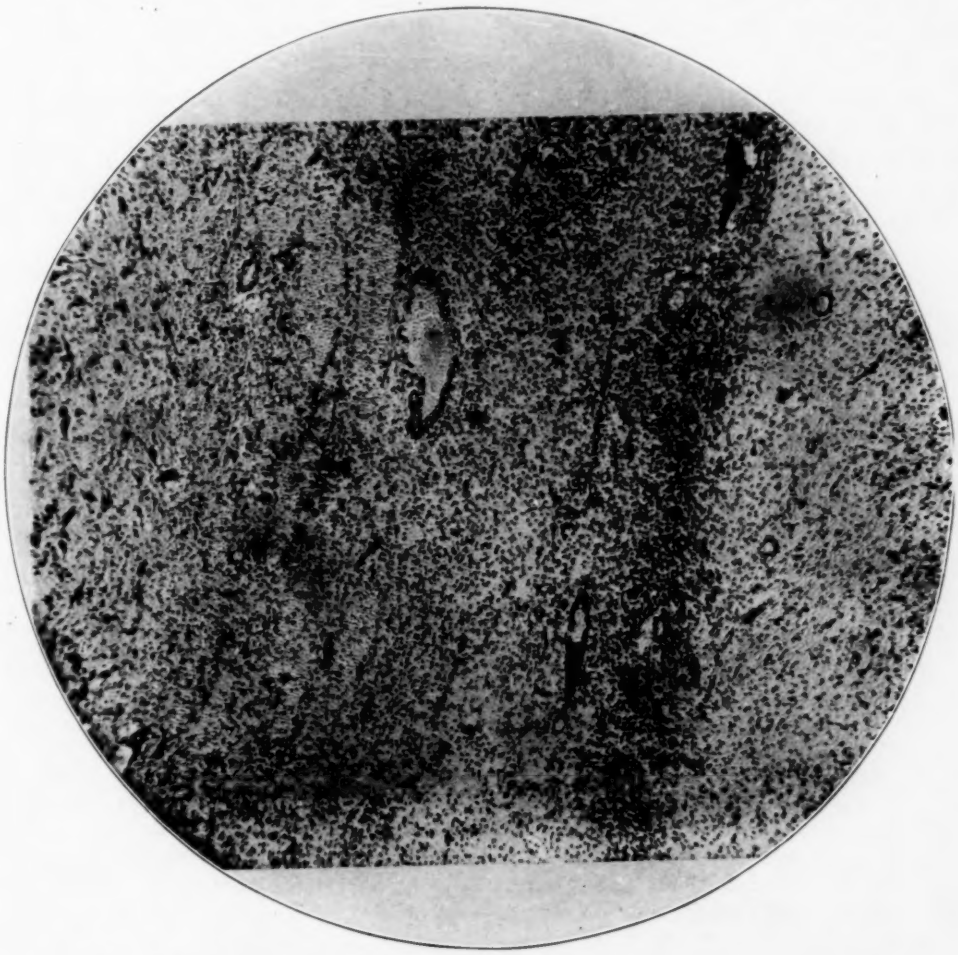

Fig. 1.—Substantia nigra (toluidin blue stain) showing the dearth of cells and perivascular infiltration.

fours caused complete absence of the torsion movements and disappearance of the opisthotonos. Necropsy was not done in this case, so the location of the lesion remains unknown. Pétren [13] and Brahme report

13. Pétren, K., and Brahme, L.: Total Immobilization in the Extremities Through Hypertonia After Epidemic Encephalitis, J. Nerv. & Ment. Dis. **57**:105 (Feb.) 1923.

two cases of total immobilization of the extremities through hypertonia following encephalitis. Their first case occurred in a boy, aged 16, who had an attack of epidemic encephalitis followed by complete immobilization of all extremities, the rigidity being so great that passive movements were impossible. The arms were held flexed at the elbow, with the hands in volar flexion; the fingers were in a claw-like position, hyperextended at the metacarpal joints, with the thumb strongly adducted and opposed. The lower extremities were fixed at the knee and hip joints and slightly flexed. In this patient, only respiration, swallowing and eye movements were possible; all passive movements were impossible. There was no evidence of involvement of the corticospinal system. Slight improvement occurred during a course of months, but the patient was left with a parkinsonian residuum. The second case reported by these authors was one of hemiplegia following encephalitis, with extreme rigidity and loss of passive motion on the paralyzed side. Musser and Bennett [14] report the case of a girl, aged 16, with extreme muscular hypertonia and catalepsy following epidemic encephalitis. The pathologic examination by Winkelman showed disease of the thalamus, and slight involvement of the corpus striatum. The substantia nigra was markedly diseased, the normal structure having almost completely disappeared, and there was perivascular infiltration in this region. This is the only case of marked rigidity in encephalitis that we have been able to find with a pathologic report demonstrating the nature and location of the lesion.

REPORT OF CASE

Parkinsonian state with extreme rigidity of all four extremities closely resembling decerebrate rigidity, with profuse perspiration. Death seven months after onset.

History.—J. S., a white man, aged 36, entered the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases in March, 1925, complaining of stiffness of the arms and legs. The family history was without significance. The past history showed that the man had been a blacksmith for twenty years, having been apprenticed at 16. He had no children. In 1905, at the age of 16, he had an attack of typhoid fever, and after an illness of a month made a complete recovery. In 1918, while in the army, a fall from a horse resulted in unconsciousness for twenty-four hours, but he was perfectly well in forty-eight hours. There were no other illnesses or accidents.

In November, 1924, while at work, the patient felt drowsy. He was able to continue at work, though not with his usual efficiency. He continued drowsy, listless and lethargic until Dec. 1, 1924, about ten days after the onset of the illness, when he had to go to bed because of fever, a feeling of weakness and because he saw double. Vision had been blurred from the first day of the illness. The double vision disappeared after two days and never reappeared, but while he was in bed the drowsiness increased; he became weaker and weaker, and slept all

14. Musser, J. H., Jr., and Bennett, A. E.: Catalepsy in Epidemic Encephalitis, New York M. J. **118**:399 (Oct.) 1923.

day but poorly at night. In January, 1925, he suddenly began to have sharp pains in both hands, most severe in the knuckles. The pain was accompanied with closing of the hand, which opened again after relaxation of pain, and was relieved by rubbing. These pains became more and more frequent, and the hands became more permanently clenched, so that about two weeks after the onset of the pains the hands were firmly clenched constantly and the patient was unable to open them.

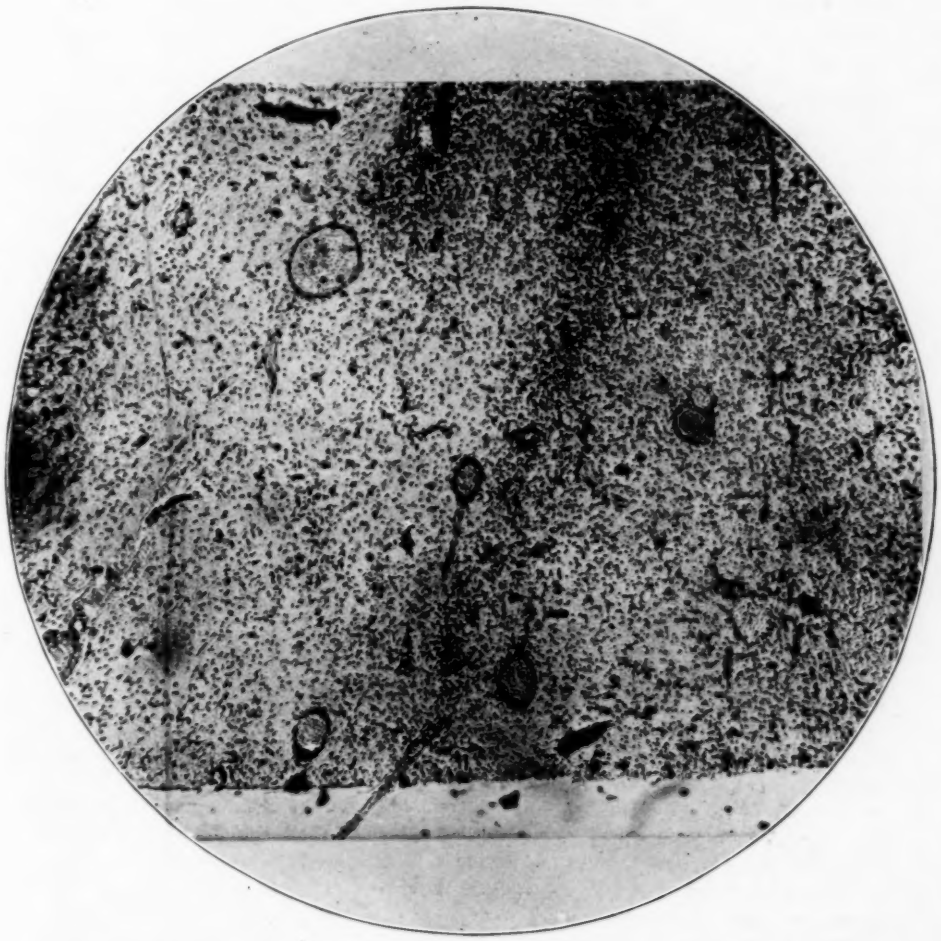

Fig. 2.—Substantia nigra (toluidin blue stain) showing the widespread perivascular infiltration.

He had no pain in the arms. About February, 1925, one month after the onset of the pains, the patient noticed stiffness of the right leg which soon became completely stiff. Pain developed in this leg, but it was described as different from that in the hands, being rather a "stiffness" than a pain. Soon after the onset of stiffness in the right leg, the left leg became stiff. Since January, 1925, the patient had noticed ptosis and profuse perspiration. There were no other relevant symptoms.

Examination.—The patient was well developed, with a mask-like expression, slow speech, monotonous voice, and extremely marked perspiration. The perspiration fell off his face, body and extremities in rivulets; it was impossible to keep him dry, and the bedclothes were constantly wet. At times the patient stated that the saliva ran as freely from his mouth as the perspiration on his body. He was constantly thirsty. He appeared emaciated and the fat over the body was scanty. The head, ears and nose revealed no abnormalities. Only a few decayed lower teeth and a few decayed upper roots were left. The gums were soft and bleeding. The left tonsil was markedly enlarged and diseased; the right appeared normal. The heart, lungs and abdomen were entirely normal. The blood pressure could not be taken because of the contractures of the arms.

The extremities showed flexion contraction of both arms, hands and fingers. There was marked adduction of both upper arms with definite pronation. The fingers were tightly flexed. The legs were held rigidly in extreme extension, with the feet strongly plantar flexed at the ankles. The legs were forcibly adducted. The arms or legs could be placed in various positions which would be well maintained for some time, gradually coming back to their former state. The pupils were irregular, the right being slightly larger than the left. There were no ocular palsies, but the ocular muscles appeared to act somewhat stiffly, motions being carried out slowly and somewhat uncertainly. The face was flat and smooth, a typical parkinsonian facies. All other cranial nerves were normal. The arm reflexes were obtainable and apparently equal but, owing to the contractures, were barely noticeable. The patellar reflexes could not be elicited because of the rigidity, and the same was true of the Achilles reflexes. The abdominal and cremasteric reflexes were present. There was no clonus or Babinski sign. In the extremities, on the motor side, active motions were not possible; at the elbow and knee joints, there were only limited passive movements, particularly on the right side. The Magnus and de Kleijn reflexes were not tested.

Laboratory Examination.—The urine showed a faint trace of albumin and occasional hyaline casts, but no other abnormalities. The red blood cells numbered 4,870,000; the white blood cells on three different occasions were: 13,000, 14,600, 13,600; the hemoglobin was 75 per cent; the blood Wassermann reaction was negative. The spinal fluid showed 1 cell, no globulin, a negative Wassermann reaction and a colloidal gold curve of 4444100000.

Course.—During the patient's stay in the hospital, about three weeks, the temperature fluctuated between 99 and 102 F.; it rose to 105 about a week before death and, after shallow fluctuations, to 107 at the time of death. He took from 150 to 250 ounces (4,440 to 7,400 cc.) of fluid a day and excreted by the kidneys only from 25 to 50 ounces (740 to 1,480 cc.). In addition to the extreme rigidity, the patient continued to show hyperpyrexia and an extreme degree of sweating. By actual timing, it was found that it required only seven minutes to wet through dry bed clothes; this condition continued to the end. The course was progressively downhill, and the patient died three weeks after entrance, or about seven months after the onset of the illness.

NECROPSY

Gross Observations.—The brain showed definite cortical atrophy, especially in the frontal lobe. There was some thickening of the pia-arachnoid, but no other abnomalities were noted.

Microscopic Observations.—The pia was fibrotic and hyperplastic but without inflammatory infiltration. The frontal cortex showed a slight disturbance of architecture, especially in the third layer, as the result of small acellular areas. Satellitosis was evident in the various cortical layers and was widespread; in

some instances neuronophagia was evident. In most of the cells the Nissl substance was absent, while in others it was granular, pale and difficult to see. Some of the ganglion cells were shrunken and contained pyknotic nuclei. The small vessels were generally thickened and hyalinized. Perivascular infiltration was not evident. The parietal cortex and the cerebellum were normal.

Caudate Nucleus: For the most part, the caudate nucleus was normal. Occasionally, however, there was involvement of the large ganglion cells, with

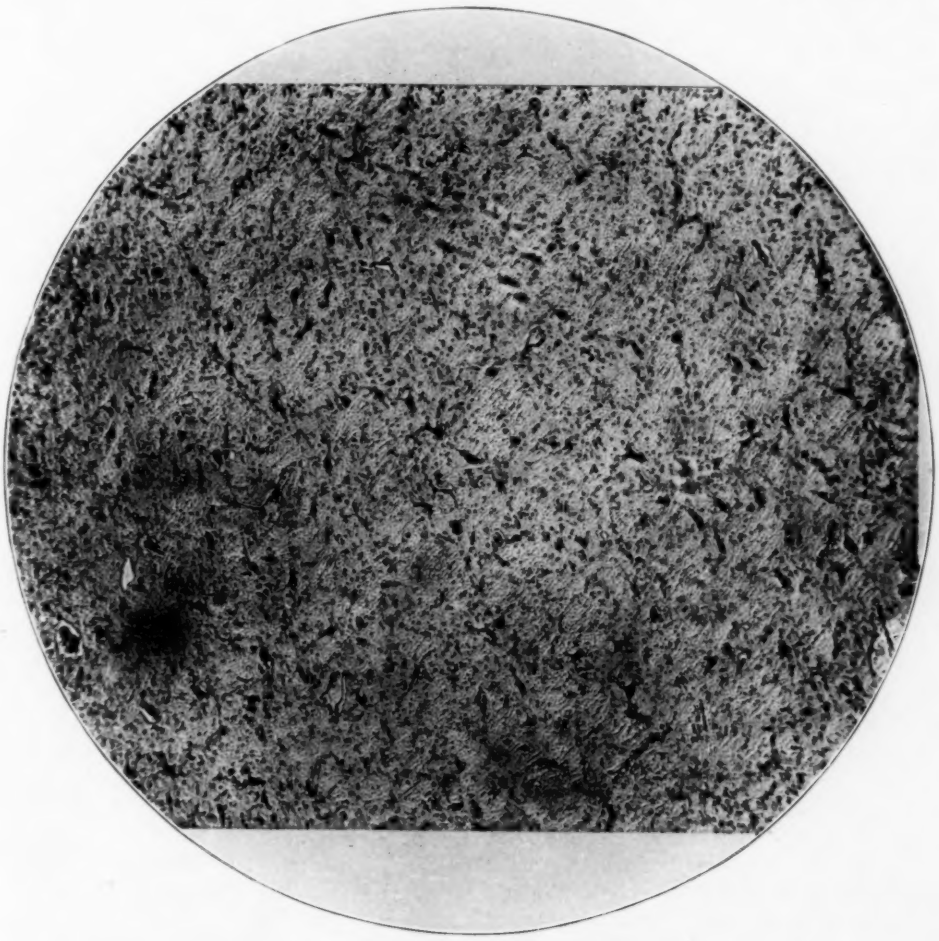

Fig. 3.—Nucleus ruber (toluidin blue stain) showing the chronic cell changes.

complete neuronophagia and formation of rosets. The small cells were, for the most part, normal with here and there formation of rosets. There was no evidence of perivascular infiltration.

Putamen: The small cells were normal. The large cells showed definite degenerative changes, and practically all of them were involved. The tigroid substance had disappeared. The outline of the cells was indefinite, the edges were ragged, and the nucleus was either pushed to one side or could not be made out at all; in some cells, globules of fat were present, and in others, glia nuclei. Sections

stained with Scharlach R showed that many ganglion cells in the putamen contained small granules of fat. The changes were within normal limits, however.

Globus Pallidus: There was no decrease in cells. Slight involvement of the ganglion cells was observed, however, some showing almost complete absence of the Nissl bodies, fatty cytoplasm, and a homogeneous structure. There was no perivascular infiltration and no increase of neuroglia. In the sections stained for fat, many of the ganglion cells in the pallidum showed fine granular deposits scattered through the cytoplasm and around the nucleus. Practically all the glia cells showed small granules of fat. By the work of Spatz[15] and Kodama,[16] fat in the cells of the pallidum has been shown to be a normal occurrence.

Thalamus: There was a fairly widespread chronic cell change, with excentric nuclei and homogeneous cytoplasm.

Substantia Nigra: Degenerative changes in the cells of the zona compacta, particularly in the medial group of cells, were present. The lateral group of cells was less affected. The cells were greatly reduced in number; those remaining were swollen and showed considerable reduction in pigment. Occasionally, active neuronophagia and the formation of rosets were present. Eccentric nucleus and homogeneous cytoplasm were common. Perivascular infiltration was present, consisting chiefly of gitter cells, though occasional lymphocytes also were present.

Nucleus Ruber: The area magnocellularis of the nucleus ruber showed definite changes in the cells. These varied from satellitosis to complete neuronophagia of the entire cell with glia roset formation. Most of the cells in this area were involved, few escaping the degenerative process. In these cells, glia cells could often be seen invading dendrons still intact, while in the cells with loss of dendrons the glia cells often could be seen in groups at the attachment of the dendron to the cell body. With loss or destruction of the dendrons, the cells had lost their triangular shape, and had become irregularly rounded, spindle-shaped or had assumed bizarre forms, depending on the degree of neuronophagia. The cytoplasm in the diseased cells was uniformly homogeneous, and in scattered cells globules of fat could be seen. The nuclei in many cases were eccentric, and occasionally showed invasion by glia cells. Some of the nuclei were pale and homogeneous, and had lost their nucleoli. Here and there cells without nuclei were distinctly seen. There was definite increase in the neuroglia. The small cells showed exactly the same changes, except that here the process was even more severe, the cells bodies being greatly swollen, the processes lost or invaded by neuroglia, and the cell itself invaded or replaced by glia cells.

Corpus Luysii: No deviation from the normal was shown.

Medulla: The various nuclei in the medulla were normal.

Spinal Cord: Examination of the cervical, thoracic and lumbar regions by the Kulschitsky method did not show degeneration in the lateral columns or, indeed, elsewhere in the cord. Weigert preparations were negative for degeneration. Anterior horn cells were practically normal.

Summary.—The observations of importance were: fibrotic thickening of the pia mater, slight cell changes in the globus pallidus and thalamus, and marked degenerative and inflammatory changes in the substantia nigra, with degenerative changes in the nucleus ruber.

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15. Spatz, H.: Ueber Stoffwechseleigentümlichkeiten in den Stammganglien, Ztschr. f. d. ges. Neurol. u. Psychiat. **78**:641, 1922.

16. Kodama, M.: Ueber den Fettgehalt des Globus Pallidus, Ztschr. f. d. ges. Neurol. u. Psychiat. **102**:237, 1926.

COMMENT

The observations and conclusions we draw are based on one case thoroughly studied both clinically and pathologically. While we are aware that it is hazardous to generalize from observations on a single case, we emphasize the fact that a typical case thoroughly studied is often of great value. Decerebrate rigidity in man is undoubtedly an unusual occurrence. While it has been reported in several instances, it is still definitely rare, and thorough pathologic investigation is uncommon. Decerebrate rigidity following epidemic encephalitis is unusual; such cases are reported as catalepsy, but few of them have the typical picture of a decerebrate preparation.

Fig. 4.—Nucleus ruber (toluidin blue stain) high power camera lucida drawing showing the cell destruction.

CASES OF DECEREBRATE RIGIDITY

The first case reported by Pétren and Brahme [13] resembled a picture of decerebrate rigidity. It occurred in a boy, aged 16, who developed complete rigidity of all four extremities following encephalitis; he recovered from this extreme rigidity but with a sequela of parkinsonism. Necropsy was not reported in this case, however, and the site of the lesion remains unknown. Turner,[17] in 1916, reported a case of decerebrate rigidity in a child, aged 18 months, who for twenty-eight days maintained an attitude of extreme rigidity in the extended position. "The feet were held in marked plantar flexion and the thighs were adducted. The upper limbs were so rigid that at times they did not rest on the bed; the forearms were persistently pronated so that the palms were turned outwards, and the fingers flexed over the thumb, which was held across the palm." Cobb [18] described a case in a child of 6½ years with extensor position of the legs and increased muscular tone. The arms were held in partial flexion at the elbow, and were stiff at the shoulders and elbows. Necropsy revealed a large tumor of the pineal body compressing the structures of the midbrain. Wilson,[19] in 1920, discussed the occurrence of decerebrate rigidity in man, and found that, from 1905 to 1920, only six cases of decerebrate rigidity in man were recorded. Most of these, however, were not true cases of extensor rigidity. Wilson reported a case in a child of 15 months who maintained a complete decerebrate attitude for six days before death. Necropsy, unfortunately, was refused. Recently, Guillain [20] reported a case in a boy of 12 who had acute encephalitis of the epidemic type followed by parkinsonism, later by disturbances in equilibration and in walking, and by torsion movements which were provoked by the entrance into action of muscles assuring the maintenance of posture. Guillain considered the rigidity in this case as of decerebrate type. He does not find Wilson's pronator sign of value, and is inclined to agree with Walshe, whose views follow. Walshe,[21] in 1923, reported the case of a woman of 23, who a year before the study complained of headaches which were followed by sudden loss of vision.

17. Turner, V.: A Case of Prolonged Hyperpyrexia in a Child with a Mid-Brain Tumor, Brit. J. Dis. Child. **13**:261, 1916.

18. Cobb, S.: Birth Palsies in Children, M. Clin. N. Amer. **4**:417, 1920.

19. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220, 1920.

20. Guillain, G.; Alajouanine, and Thévenard: Attitude d'extension et de torsion dans un cas d'hypertonie diffuse post-encéphalitique à début parkinsonien—rapports avec la rigidité décérebrée — troubles du tonus de l'equilibre, Rev. neurol. **2**:303 (Aug.) 1925.

21. Walshe, F. M. R.; A Case of Complete Decerebrate Rigidity in Man, Lancet **2**:644, 1923.

There were bilateral papilledema and bilateral Babinski sign, with increased tendon reflexes. After subtemporal decompression a left hemiplegia developed, followed in four weeks by a bilateral hemiplegia and an inability to swallow. At necropsy, a suprapituitary cyst was found with calcareous deposits which produced decerebration by "com-

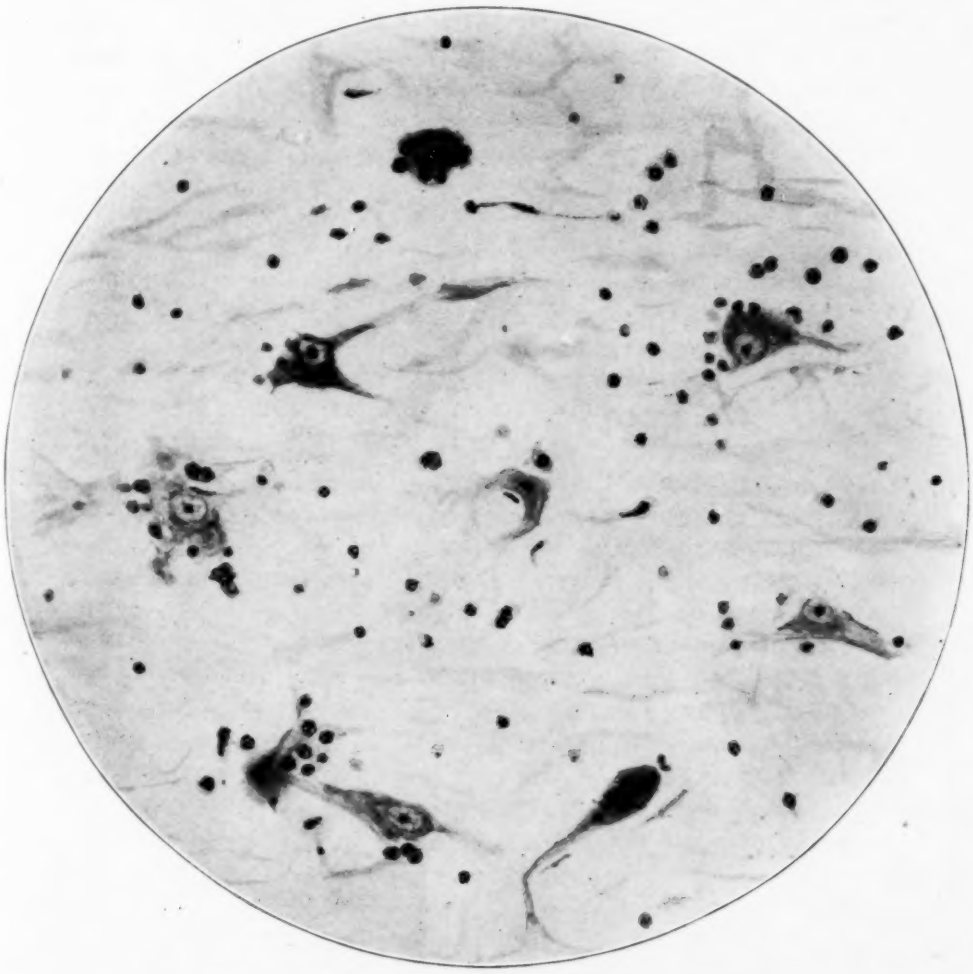

Fig. 5.—Nucleus ruber (toluidin blue) high power camera lucida drawing showing the cell destruction.

pressing the structures in the anterior part of the brain stem and by cutting off the cerebral hemispheres from functional connection with lower levels." Walshe considers this case an example of true decerebrate rigidity. His patient lay with arms semiflexed at the elbows, forearms pronated, wrist and fingers flexed, and legs extended and

adducted. The rigidity was plastic and "clasp-knife" in nature. In this case there was undoubtedly involvement of the corticospinal system. A similar case was reported in 1925 by Davis.[22] It occurred in a boy, aged 4, whose history resulted in a diagnosis of a suprasellar lesion. He was unable to move the upper or lower extremities, all of which were definitely spastic. The legs were in strong adduction and extension, and the feet in plantar flexion. The arms were held in semiflexion at the elbows and were adducted. There were exaggerated tendon reflexes and bilateral Babinski sign. Operation, with partial evacuation of a suprasellar cyst, was followed by increase in the rigidity in all four extremities. Necropsy revealed a large suprasellar cyst. The motor cells in the cortex showed definite retrograde changes; those of the midbrain, chromatolysis. Changes were not found in the basal ganglia. In the last two cases, those of Walshe and Davis, there was undoubted involvement of both corticospinal tracts, and while the patients may have presented the appearance of a decerebrate preparation, the conditions were really bilateral hemiplegias.

THEORETICAL INTERPRETATION OF DECEREBRATE RIGIDITY IN MAN

The interpretation of the reason for the decerebrate picture in man has not been uniform by any means. Wilson[19] divided the cases of decerebrate rigidity in man into two groups: those occurring in unconscious states, and those in conscious life. The former group he further subdivided into cases with and without tonic fits in addition to the extensor phenomena. Walshe[23] differs from other authors in that he insists that the decerebrate rigidity seen in extrapyramidal disease is not of the same type as that produced in experimental animals. He is decidely of the opinion that "muscular rigidity comparable with experimentally produced decerebrate rigidity is most commonly seen in man in association with hemiplegia, with cerebral diplegia of pure pyramidal type, and in paraplegia in extension from spinal cord lesions. . . . It follows from these considerations that all forms of extrapyramidal motor disease associated with muscular rigidity, with or without involuntary movements, must be rejected as having no relation with decerebrate rigidity. In such cases, the quality of the rigidity and its incidence, and the absence of the essential interference with the functions of the pyramidal system clearly indicate that we are dealing with a fundamentally different set of phenomena." While the type of

22. Davis, L. E.: Decerebrate Rigidity in Man, Arch. Neurol. & Psychiat. **13**:569 (May) 1925.

23. Walshe, F. M. R.: The Decerebrate Rigidity of Sherrington in Man, Arch. Neurol. & Psychiat. **10**:1 (July) 1923.

rigidity seen in hemiplegia, with its plasticity, its lengthening and shortening reactions and its "step-ladder" phenomenon, corresponds to what is seen in the decerebrate animal, certainly the lesion in such cases is far from corresponding with the experimental site. Cobb [18] outlines clearly the various impulses playing on the anterior horn cells which can produce decerebrate pictures. There is first the corticospinal path which, if injured above the striatum, causes loss of voluntary motion. Then there are the paths from the red nucleus and the vestibular nuclei which, when acting without cortical and striatal control, play unhampered on the anterior horn cells and give the phenomena of decerebrate rigidity.

That the case which we report is one of decerebrate rigidity in man can hardly be questioned. The attitude of the patient, the type of rigidity, and the freedom from involvement of the corticospinal system indicate a picture of extensor rigidity similar to that found in the experimental animal. The legs were held in extreme extension and adduction, with the feet plantar flexed. This attitude of the lower extremities seems to be a constant feature of all the cases reported as decerebrate rigidity. The one feature in our case that is not in accord with a picture of true extensor rigidity, as it is ordinarily conceived, is the flexion contraction of the upper extremities. This may be justified as an integral part of a chronic decerebrate picture from several points of view. In the first place, the studies of Bazett and Penfield [24] on chronic decerebrate animals which were kept alive for two weeks or more seem to show definitely that flexion may, and often does, occur in the decerebrate animal. These investigators demonstrated that flexed positions in the chronic animals are comparatively common, indeed, that some degree of flexion is usually found in one or more of the limbs after the first five to ten days, and though it may alternate with extension, yet flexion may sometimes be the more constantly maintained posture. "The most common of these flexed positions is a rigid semi-flexion of the forelimbs, or of one forelimb, with an acute rigid flexion of the ankle joints and extension of the claws of the forelimbs." From this experimental evidence it seems possible, therefore, to obtain flexion attitudes in the chronic decerebrate preparation, and the attitude of flexion rigidity of the upper extremities in our case need not necessarily militate against the picture of decerebrate rigidity. From another angle one might state that many of the cases of decerebrate rigidity reported in the literature have shown flexion rigidity in the upper extremities. The cases of Walshe and Davis, together with a case of Wilson, fall in this category. Walshe [21] accounts for the

24. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrate Animal in the Chronic as Well as the Acute Condition, Brain **45**:185, 1922.

flexion attitude in the upper extremities, which he considers ancillary to the picture by the fact that in man the forelimbs are no longer simple locomotor props but have taken on new and highly complex functions under the influence of the cerebral cortex. In consequence, we should not expect them to take up the extensor attitude of standing. Turner's case, however, showed extensor rigidity in all four extremities. Wilson points out the occurrence of such flexor attitudes, and compares them with the tetanus-like seizures of Hughlings Jackson, ascribing their occurrence to a cerebellar influence. Another constant feature of these clinical cases of decerebrate rigidity is the occurrence of pronation in the upper extremities. In practically every instance, whether the arms are flexed or extended, there is some degree of pronation of the forearm on the arm. This is as constant as the extension of the lower extremities with plantar flexion.

A close study of our case reveals the fact that the lesion was sharply localized. The corticospinal systems were entirely intact; the caudate nucleus and globus pallidus were affected, little, if at all, and the putamen was moderately involved. All the pathologic changes in the case center around the substantia nigra and the nucleus ruber. In the substantia nigra were definite subacute and chronic encephalitic changes with perivascular infiltration with phagocytic cells, and with practical destruction of the mesial group of cells. The lateral group of cells was relatively intact. The nucleus ruber throughout had marked evidence of neuronophagia and chromatolysis, and slight evidence of perivascular infiltration.

How can these observations be explained and correlated not only with the clinical symptoms, but also with the numerous views concerning the nature of decerebrate rigidity? In the first place, it must not be forgotten that in addition to the extreme rigidity in our case, there was also a parkinsonian syndrome, as shown by the masklike facies, the sialorrhea and other characteristic symptoms. It is possible that this part of the clinical picture was dependent on the lesions in the substantia nigra, in accord with the views of Trétiakoff [25] et al. To the lesion in the nucleus ruber we may ascribe the responsibility for the extreme rigidity, and in this our case is in accord with those observations of Weed [26] and Graham Brown,[27] and with the views of Wilson. Weed, for example, considers the mesencephalon, especially the nucleus ruber, of prime importance in the production of decerebrate rigidity. Graham Brown is of the same opinion, while Sherrington, in a personal

25. Trétiakoff, C.: Rev. neurol. **27**:954, 1920.

26. Weed, L. H.: Observations on Decerebrate Rigidity, J. Physiol. **48**:205, 1914.

27. Brown, Graham: On Postural and Nonpostural Activities of the Mid-Brain, Proc. Roy. Soc. Med. **87**:145, 1913.

letter to Wilson, considers the nucleus ruber of "capital importance." Wilson expresses his views with regard to the matter as follows: "From the point of view of experimentation then, the conclusion seems clear enough that the withdrawal of cortical control, by decerebration, from the spinal motor center allows them to be played on by a non-pyramidal motor influence of a tonic nature, or rather allows this doubtless constantly tonizing factor to reveal itself in action. The center of this attitude- or posture-producing motor activity is apparently the nucleus ruber and not the cerebellar nuclei."

It would hardly seem justifiable on our part to elaborate a theory on the basis of a single observation, but in our own case, granting the part of the substantia nigra in the production of the parkinsonian picture, it would then seem that the rôle of the nucleus ruber was essentially as stated by Wilson, and that we may look on the decerebrate picture produced as essentially a release phenomenon. On the other hand, if we interpret the views of Wilson correctly, the rôle of the nucleus ruber consists in the maintenance of a continual tonizing effect on the cells of the anterior horn, thus being responsible for the maintenance of a certain degree of tone. From the recent work of Rademaker,[28] however, it would seem that the rôle of this nucleus consists of the control and distribution of tone and thus inhibits extensor rigidity. A lesion therein should, therefore, produce a condition of this sort, in the nature of a release phenomenon. This would seem to be the case in the patient whom we have studied, but of course the influence of the substantia nigra must not be overlooked. The lesion in the nucleus ruber leaves the vestibular systems to act on the anterior horn cells without control, and in this way causes the evidences of postural tone in our case.

CONCLUSIONS

1. A case of decerebrate rigidity in man following encephalitis is presented.

2. Pathologically, inflammatory and degenerative changes were found in the substantia nigra, and degenerative changes chiefly, with some inflammatory signs, in the nucleus ruber.

3. The pathologic observations coincide with the views of Weed, Brown, and Wilson in attributing to destructive lesions in the nucleus ruber an important rôle in the production of decerebrate rigidity.

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28. Rademaker, G. G. J.: Die Bedeutung der rote Kerne und das übrigen Mittelhirns für Muskeltonus, Körperstellung und Labyrinthreflexe, Berlin, J. Springer, 1926.

INTRADURAL SPINAL LIPOMA

REPORT OF A CASE AND SYMPTOMS FOR TEN YEARS IN A CHILD AGED ELEVEN; REVIEW OF THE LITERATURE *

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Lipoma of the spinal cord unassociated with spina bifida, extradural or intradural, is rare. In a review of the literature up to 1926, reports of only nine extradural and nine intradural lipomas have been found. Their rarity is further indicated by the fact that of 35,000 necropsies reviewed by Schlesinger,[1] 6,540 showed some form of tumor, but no intradural spinal lipoma was found. In the statistics of the New York Neurological Institute, no instance of either extradural or intradural lipoma unassociated with spina bifida was found at either necropsy or operation. Schlesinger's review of the literature brought forth reports of three intradural and twelve extradural lipomas. Spiller, in 1899, reported a case of a minute intradural lipoma in the filum terminale and verified the cases reviewed by Schlesinger without finding a single additional one. He was unable to find one reference (Holmes, 1885) cited by Schlesinger, nor have I been more successful. There seems to have been some confusion concerning Strümpell's case,[2] which Schlesinger cites in one place as intradural tumor and in another as extradural tumor. Since the original reference was not available to Spiller when he reported his case, Strümpell's was included as an intradural tumor, whereas in reality it was extradural. Three of the tumors included in Schlesinger's review and accepted by Spiller as extradural lipomas without spina bifida apparently were associated with spina bifida, and hence are not included in this series. Omission of these reduces the number of extradural and intradural tumors in Spiller's report to twelve—nine extradural and three intradural. Ritter, in 1920, found no additional cases of either extradural or intradural lipomas not previously reported by Schlesinger and Spiller.

Lipomas with spina bifida, so-called "sacral tumors," as pointed out by Chiari and von Recklinghausen, are not uncommon, but their clinical manifestations and history are so unlike those of uncomplicated lipomas that they have not been included in this series.

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* From the Neurosurgical Service, Neurological Institute.

1. Schlesinger, Herman: Beitr. z. Klin. d. Rückenmarks- u. Wirbeltumoren, Jena, 1898.

2. Strümpell: Ein extradurales Lipom, Arch. f. Psychiat. **29**:1004, 1897.

Lipomas within the cerebral meninges are much more common than those within the spinal meninges. Sury and Zuckermann reviewed the literature over a period of fifty years, and collected reports of approximately fifty cases of intradural cerebral lipomas. In a review of the literature of about the same period, reports of only three intradural spinal lipomas were found. Intracranial lipomas are most commonly found in the region of the corpus callosum, the ventricular ependyma and the base of the brain. Lipomas in the region of the corpus callosum were described by Kirkbride,[3] de Steiger,[4] Ernst,[5] Bostroem,[6] Naciarone,[7] della Rovere [8] and others. Small lipomas of the corpora quadrigemina were found by Taubner [9] and Bernhardt.[10] While the majority of intracranial lipomas are small, a few large ones have been reported. The large tumors are more likely to be mixed tumors, such as pial epidermoid, pial dermoid and pial lipomas, which are believed by Bostroem to develop from embryologic inclusions at the time of the formation of the secondary brain vesicles.

ORIGIN

The appearance of an intradural spinal lipoma in tissues generally considered free from fat cells is of special interest. There are two views concerning their origin. The first is that occasional fat cells occur in the pia and that the tumor arises from these cells—a new growth from preexisting tissue (Virchow). Though the pia-arachnoid is said to be devoid of fat, occasional fat cells have been found by Chiari, Kölliker, Bostroem and others. In a systematic search of the cerebral pia-arachnoid, Chiari found occasional fat cells scattered here and there. It is true that fat cells are rarely found in the pia-arachnoid; intradural lipomas are also extremely rare. The second view is that intradural lipomas are due to inclusions of embryologic rests within the meninges at the time of the formation of the neural tube (Kölliker,

3. Kirkbride, T.: Lipoma of the Brain, Orths Festschr., Berlin, 1903, p. 515.

4. De Steiger, A.: Two Cases of Lipoma of the Brain, J. Ment. Sc. **48**:64, 1902.

5. Ernst, P.: Ein bügelförmiges, gemischtes Lipom auf dem Balken, Zeigler's Beitr. (supp.) **7**:1, 1905.

6. Bostroem, E.: Ueber die pialen Epidermoid, Dermoid und Lipome, Zentralbl. f. allg. Path. **8**:1, 1897.

7. Naciarone, A.: Ein seltener Fall von Lipom der Gehirnhaüte, Gior. med., 1904.

8. Della Rovere, D.: Due casi de lipomi della pia meningo, Clin. med. ital. **41**:129, 1902.

9. Taubner: Zur Casuistik und Entwickelung der Hirnlipome, Virchows Arch. f. path. Anat. **110**:95, 1887.

10. Bernhardt, M.: Beitr. z. Symptomatol. u. Diagnostik d. Hirngeschwülste, Berlin, 1881.

Bostroem, Ernst and others). Bostroem believes that skin anlage may be carried in at the time of the formation of the secondary brain vesicles, and this inclusion of whole skin explains the presence of intracranial dermoids, epidermoids and mixed lipomas. Since intracranial lipomas are often mixed tumors, Ernst feels that they are to be explained only on the basis of embryologic inclusions; della Rovere, however, holds that intracranial lipomas are due to mesodermal and not to epidermal inclusions. Ritter [11] believes that a part of the cutis plate or even part of the sclerotome may be included at the time of fusion of the neural fold in the midline to form the neural tube. Since Ritter's patient had an associated congenital kyphosis and scoliosis, he felt that this defect in the bony structures might be due to congenital anomalies in the sclerotome. Hence, congenital defects in the formation of the neural tube may have associated with them abnormalities in the mesodermal tissues about the neural tube. Lipomas in general are frequently associated with congenital anomalies, especially, as has been said, with spina bifida. In addition to a congenital kyphosis and scoliosis found by Ritter, Wolbach and Millet's case [12] showed a congenital cleft palate, a harelip, and absence of one kidney and ureter; in my case there were bilateral cervical ribs. The last mentioned condition is frequently associated with defective development of the spinal cord, and especially with syringomyelia.

The histologic structure of Ritter's intradural spinal lipoma gave no evidence of its origin from either the cutis plate or the sclerotome. In this connection it is interesting to note that in all of the intradural spinal lipomas, with one exception, pure and not mixed lipomas were found. As a coincidence, at necropsy on a tabetic patient, Gowers [13] found a small fatty tumor overlying the conus; it was a myolipoma, containing numerous bundles of striated muscle. Since this is the only mixed tumor in the series of intradural spinal lipomas, one might be tempted to consider it a different origin. From its position in the lumbosacral region, it is possible that it may have been some form of occult spina bifida with an associated myolipoma, since in this region mixed lipomas are common.

A myolipoma associated with spina bifida has been reported; it grew into the spinal cord in the vicinity of the conus and completely severed the conus from the rest of the cord. Whatever the source of Gowers' mixed tumor, the remaining intradural spinal lipomas were not mixed tumors and gave no evidence of origin from either epidermal or dermal inclusions. The fact that intradural lipomas may be congenital speaks

11. Ritter, A.: Ein Lipom der Meningen des Cervicalmarks, Deutsche Ztschr. f. Chir. **152**:189, 1920.

12. Wolbach, S. B., and Millet, J. A. P.: Diffuse Subdural Lipomatosis of the Spinal Cord in an Infant, Boston M. & S. J. **168**:681, 1913.

13. Gowers: Myolipoma of the Spinal Cord, Tr. Path. Soc., London, 1876, p. 27.

somewhat for the theory of their derivation from embryologic rests. Birsch and Hirschfeld found a pial lipoma in a new-born infant, and Parrot, one in a girl, aged 3. Morestin found a congenital lipoma in the gastrocnemius, and Vickery a congenital lipoma weighing 12½ ounces (354.3 Gm.) arising from the left sciatic nerve.

Heterotopic lipomas have been described in numerous locations, such as the intestines, uterus and kidney. Those found in the kidney are thought to be due to inclusions of fat cells from the kidney capsule. As far as the histologic structure of the tumor in the case here reported is concerned, no data are at hand from which to determine whether the tumor is derived from inclusions or from preexisting fat cells in the pia-arachnoid, since the tumor is made up of fat cells only and contains no other tissues. Had the tissue been obtained at necropsy together with the spinal cord, instead of at operation and without the surrounding tissue, such evidence as to its source might be available.

It is difficult to understand how mesodermal tissue can be enfolded within the neural tube, since the neural plate is formed by proliferations of preexisting ectodermal cells forming folds at the neurosomatic junction. These folds soon fuse in the midline to form the roof plate, which is covered immediately after fusion by the somatic ectoderm before any mesenchyme intervenes. Mesodermal tissue is found between the neural and the somatic ectoderm only a long time after fusion, and hence could not have been included at the point of fusion, unless the fusion of the neural tube was incomplete when the somatic ectoderm fused across the neural tube. We know that somatic ectoderm may grow over and cover the neural tube even though the neural tube itself has not closed. This is seen in the large clefts found in both the neural and the mesodermal elements in spina bifida. These have complete skin covering even though the neural tube has not closed. It is possible that mesenchyme may grow into such a cleft in a neural tube, which later may or may not close. Any inclusion of mesodermal tissue at this stage might readily be the source of an intradural lipoma as well as of the lipomas so frequently associated with the more obvious closure defects in spina bifida, where they fill in the cleft, not only of the neural tube but even of the vertebral column.

However, another possible source of intradural lipomas may be the mesenchyme which penetrates into the neural wall to lay the foundation for the vascularization of the central nervous system. Mesodermal tissues thus carried into the neural tube might be the source of such an intramedullary lipoma as was found by Kölliker in the lumbar enlargement of the cat, or that found by Turner [14] as an intramedullary lipoma

14. Turner, C.: Lipomatous Tumor? (Sarcoma) of the Spinal Cord, Tr. Path. Soc., London **39**:25, 1888.

of the midthoracic region in a woman. The point then arises: Are not the intradural lipomas, which apparently invade the spinal cord, primarily intramedullary inclusion lipomas which secondarily grow out of the cord? These tumors have a capsule over the extramedullary portion formed by the pia-arachnoid, but no capsule has been shown to

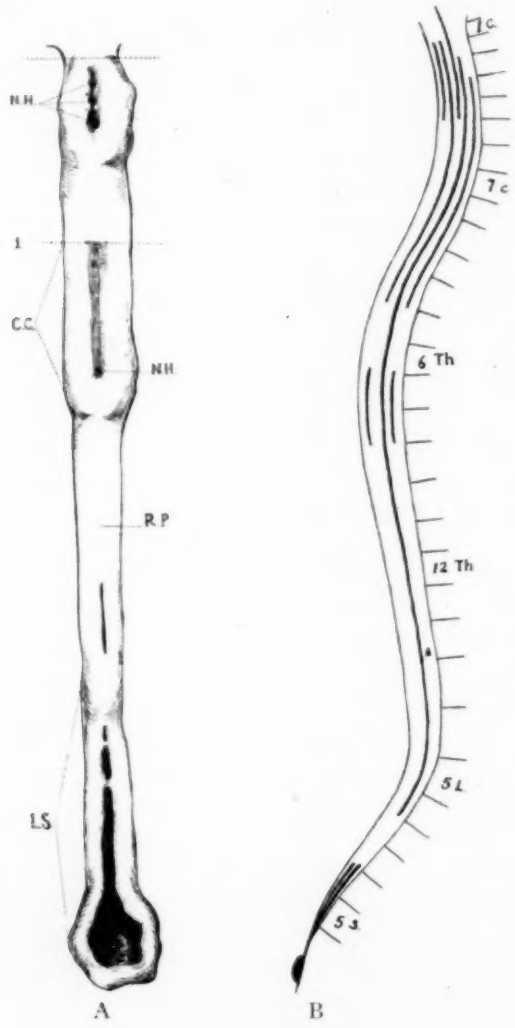

Fig. 1.—*A*, schematic drawing of reconstruction of neural tube from 12 somite embryo (Schulte and Tilney). *N. H.* indicates neural hiatus; *1*, transition between brain and spinal cord; *C. C.*, cervical cord; *R. P.*, roof plate; *L. S.*, lumbosacral cord. *B*, schematic drawing to show segmental distribution of intradural spinal lipomas. The tumors are indicated by hatched lines, except that of the filum terminale, which is indicated by a black area.

separate the tumor within the cord from the cord tissues. Invasion of tissue by a lipoma is unusual; hence, it may well be that the intradural lipomas primarily are intramedullary tumors and only secondarily have become extramedullary. If the theory of inclusion of embryologic rests at the time of fusion of the neural tube is accepted, it is possible for the inclusion to be carried into the neural wall itself. The two regions of the spinal cord in which lipomas predominate are the cephalic and caudal extremities. In the latter positions the neural tube is late in closing, the roof plate having covered over the intervening portion earlier. In these two situations, inclusions of mesenchyme might be carried into the wall of the neural tube and give rise to intramedullary lipomas.

From a theoretic standpoint, still another view may be considered; namely, that the primitive tendency of the spongioblast and ependymal cells of the central nervous system to accumulate fat and form myelin may be continued abnormally both in amount and in duration.

According to Wlassak,[15] as Tilney and Casamajor pointed out in their studies of myelogeny,[16] myelin is first deposited in the primitive embryonic nervous system in the protoplasm of the spongioblast, in the ependymal cells and the epithelial cells of the third and fourth ventricles. The connective tissue cells of the pia are the first to show accumulation of myelin. Later, myelin is accumulated in the spongioblast and ependymal cells. It is brought to the central nervous system through the blood vessels of the pia, which are nearly enclosed in myelin on the side directed toward the nervous system. The epithelial cells of the roof plate of the third and fourth ventricles continue to accumulate myelin in postembryonic life in lower animals. According to Adami and Aschoff,[17] myelin is an oleic acid compound having a double refraction characteristic of a fatty substance. It is possible that the tendency of such cells as the spongioblasts and the ependymal cells to accumulate myelin is continued as a perverted function, and that large abnormal deposits of myelin might then occur. Theoretically, this might be the original source of the intradural collections of fat seen within both the spinal cord and the brain. It is interesting in this connection to observe that small cerebral lipomas without clinical signs are frequently found in the ependymal cells and in the region of the third ventricle. In these

15. Wlassak, R.: Die Herkunft des Myelins, Beitr. z. Physiol. d. nervösin Stutzgeweber **6**:453, 1898.

16. Tilney, Frederick; and Casamajor, Louis: Myelinogeny as Applied to the Study of Behavior, Arch. Neurol. & Psychiat. **12**:1 (July) 1924.

17. Adami, J. G.: The Myelins and Potential Fluid Crystalline Bodies of the Organism, J. A. M. A. **48**:462 (Feb. 9) 1907; Adami, J. G., and Aschoff, L.: On the Myelins, Myelin Bodies and Potential Fluid Crystals of the Organism, Proc. Roy. Soc., London, series B, **78**:359, 1906.

regions and in the vicinity of the corpus callosum, these tumors appear as little more than simple fatty deposits which might well be thought of as a continuation of the tendency to accumulation of fat, which is a function of certain cells of the central nervous system. The histologic appearance of the lipomas described by Kölliker in the lumbar enlargement of the cord in the cat seems to be little more than a collection of fat cells similar to that seen in the region of the corpus callosum, which have been described as cerebral lipomas. However, the histologic study of the tissues of intradural spinal lipomas show them to be all rather typical fatty tumors having the usual histologic structure found in lipomas irrespective of their source. No evidence of spongioblasts or ependymal cells has been demonstrable. Moreover, lipomas consist of fat and not of myelin.

EXTRADURAL SPINAL LIPOMAS

The first extradural spinal lipoma is that described by Chapelle [18] (1847) in a child, aged 4, who had had symptoms for a year and a half. Up to 2½ years, the child had played with other children and apparently had been in good health. Then weakness was noted, and the child died at the age of 4, of scarlet fever. At necropsy the dura was found surrounded by extradural fat from the cervical region to the caudal end of the cord, mostly on the dorsal aspect. However, toward the caudal end of the cord the fatty mass became larger and in the region of the conus and cauda compressed the cord and nerve roots. The fat was made up of rather large lobules, having the same color, consistency and reactions as ordinary fat.

Albers,[19] in his atlas (1847), illustrated a spinal dura with multiple lipomas on its outer surface. Obré,[20] in 1852, found a localized fatty tumor in a child, aged 3, who at the age of 1 had begun to have convulsions. Following the first convulsion the child was paralyzed, especially in the left leg. Similar convulsions recurred, and, at the age of 3, the child died in a convulsion. Examination of the brain and meninges showed no abnormality, but beneath the upper thoracic vertebrae was a fat deposit between the dura and vertebrae, measuring 2.5 by 1 cm. This mass compressed the cord. The tumor consisted of fat cells which seemed to contain fat in a more solid form than that usually seen in ordinary fat tissue.

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18. Chapelle: Extradurales Lipom, Bull. de la Soc. anat. **22**:6, 1847.

19. Albers: Extradural Lipoma, Atlas der path. Anat. f. prakt. Aerzte, Bonn, 1847.

20. Obré: Lipoma of the Meninges, Tr. Path. Soc., London **3**:248, 1852.

Virchow[21] (1857) described a case of multiple fatty tumors in a man, aged 53, who eleven weeks before death complained of unbearable pain in the lumbar region, paresis of both lower extremities, incontinence of bladder and rectum, and also decubitus. A fatty tumor connected with the arachnoid was found in the cerebral fossa, and another in the vertebral canal in the lumbar region, measuring 3 by 1.5 cm. Virchow considered these neoplasms a peculiar and special type of lipoma and named them myxoides lipomatodes.

Hoffmann,[22] in 1869, described a woman, aged 43, who nine months previously had begun to show signs of spinal cord disease. At necropsy a glioma of the cord was found, and numerous fatty tumors on the outer surface of the dura, extending from the seventh thoracic to the first sacral vertebra, the largest of which was in the region of the fifth lumbar and first sacral vertebrae. The bones here were described as soft and pliable. Similar fatty tumors had grown into the dorsal nerve roots. It would seem to be a question whether this was not an incomplete spina bifida, with the bony defect covered over by incomplete bone formation. It is noteworthy that associated with the lipoma a glioma of the spinal cord was found, though not immediately beneath the lipoma and apparently unconnected with it.

Berenbruch,[23] in 1890, described a patient, aged 16, who presented a large congenital tumor on the back, which had gradually become of tremendous size. He had pain in the legs and weakness in walking. About two months later, the large superficial congenital lipoma was removed; the patient died a short time after the operation. At necropsy numerous tumors were found: a congenital lipoma over the left side of the back; a fatty tumor containing numerous blood vessels in the right scapular region; an angiolipoma of the right kidney capsule; an extradural spinal tumor, mostly angioma, with here and there a few fat cells, and within the spinal cord a pure angioma. The patient thus showed all gradations from pure lipoma to pure angioma.

Starr[24] (1895) described a patient, aged 50, who four months previously had had pain in the right side of the abdomen, extending to the opposite side, with signs of spinal cord compression. At operation, a small fatty tumor, lying extradurally and measuring ½ by ¾ inch (13 by 19 mm.) was found beneath the arch of the tenth thoracic

21. Virchow, R.: Ein Fall von bösartigen, zum Theil in der form des Neurons auftretenden Fettgeschwülsten, Virchows Arch. f. path. Anat. **11**:281, 1857.

22. Hoffmann, E. K.: Beiträge z. Kenntniss des Geschwülsten der Gehirns und Rückenmarks, Ztschr. f. rationalle Med. **34**:189, 1869.

23. Berenbruch, K.: Ein fall von multiplen Angio-Lipomen kombiniert mit einem Angiom des Rückenmarks, Tübingen, 1890.

24. Starr, Allen: A Contribution to the Subject of Tumors of the Spinal Cord, Am. J. M. Sc. **109**:622, 1895.

vertebra, and another beneath the arch of the eleventh thoracic vertebra. No histologic examination was reported. The patient did not improve, and a second operation was performed at a higher level. A tuberculous abscess which had pointed laterally was opened, and the vertebrae were said to be involved. Following the second operation, the spinal cord signs improved. Starr was doubtful whether the symptoms were due to the fatty deposits beneath the tenth and eleventh thoracic vertebrae. I doubt that this was a true fatty tumor, for extradural fat deposits of the size described in this patient are normal.

Liebscher [25] (1901) found an angiolipoma in a woman, aged 56, who four and a half years previously had had girdle signs and a feeling as if walking on sand. The tumor measured 7 by 2 cm. and was situated between the dura and the ventral surface of the vertebrae; it was adherent to the periosteum, but only loosely connected with the dura. The tumor was dark red and highly vascular, and at the level of the seventh and eighth thoracic segments the cord was markedly compressed, being only 3 mm. thick.

Strümpell,[2] in 1897, presented a case of an extradural lipoma compressing the cord, no details of the case being recorded.

Three reports, by Johnson,[26] Témoin,[27] and Zavaleta and Masi,[28] have been included in previous references among extradural lipomas without spina bifida. They do not belong to this group, I believe, but should be included in the group of sacral tumors associated with spina bifida, with which this study is not concerned.

Johnson found in an infant, aged 10 months, a tumor over the sacrum about the size of an orange, which was connected with the meninges through an opening in the dorsal wall of the sacral canal. The mother stated that the infant was born with an ulcer over the sacrum and a swelling beneath the ulcer. This swelling remained about the same size for a few months and then enlarged until it was approximately the size of an orange. Témoin described a sacral tumor weighing 900 Gm. in an infant, aged 9 months, in whom there were no motor signs or other evidences of spinal cord disturbance. The tumor was connected with a pedicle within the vertebral canal, and a small opening large enough to admit the little finger was found between the vertebrae. This cannot be included as an extradural tumor, since there were no

25. Liebscher, C.: Angiolipom des Wirbelkanals mit Kompression des Rückenmarks, Prag. med. Wchnschr. **26**:189, 1901.

26. Johnson, A.: A Fatty Tumor from the Sacrum of a Child, Connected with Spinal Membrane, Tr. Path. Soc., London **8**:16.

27. Témoin: Lipome périméninge simulant un spina bifida, Arch. prov. de chir. **1**:179, 1892.

28. Zavaleta and Masi: Lipome de l'archnoide rachidienne, in Chipault's étude de chirurgie médullaire, Paris, Alcan, 1893.

neurologic signs, and the tumor mass was almost exclusively superficial. Zavaleta and Masi, in 1893, described a lipoma in an infant, aged 2 months, in the region of the fourth lumbar vertebra. The tumor was about 10 cm. in diameter; it was attached to the arachnoid by a pedicle and was covered by the dura at its base.

It will be noted that, in extradural tumors not associated with spina bifida, considerable variation exists in the tumor type. Multiple lipomas were found in five instances, mixed tumors in three, angiolipoma in two and lipoma myxoides in one. In two instances other neoplasms of the spinal cord were found: a glioma, not immediately beneath the fatty tumor, and a cavernous angioma immediately beneath, but not connected with, an extradural angiolipoma.

Ebinger,[29] in 1901, found an extradural lipoma in the vertebral canal of a cow, in the region of the third and fourth lumbar vertebrae. The tumor was a pure lipoma measuring 8 by 2 cm., lying dorsal to the dura and adherent to the periosteum and compressing the spinal cord. The cow had marked symptoms of spinal cord disease. Since a relatively thick epidural fat pad is usually found, one might expect extradural lipomas to be more frequent in this region, and certainly more frequent than intradural lipomas.

INTRADURAL LIPOMAS

The first case was described by Gowers[13] in a tabetic patient, in whom a fatty tumor attached to and arising from the pia was found overlying the right half of the conus. The tumor was ½ by ½ by ⅜ inch (13 by 13 by 9 mm.), and was crescentic in shape with the nerve roots of the cauda equina running through it. It was composed of oval fat cells, resembling closely the cells of ordinary fat tissue. The tumor was enclosed in a definite capsule identical with the pia. In addition to ordinary fat cells, many striated muscle fibers were seen.

The second case, described by Turner,[14] was that of an obese woman, aged 54, who for a period of twelve years had had gradually increasing paralysis of both lower extremities. At necropsy a lipoma in the middle thoracic cord was found. It was described as about the size of a large filbert, forming a well defined fusiform swelling in the substance of the cord. It had pushed the cord substance to the right and had almost wholly infiltrated and destroyed the cord. While Turner found nothing histologically to differentiate this from typical fatty tissue, he felt that because it had invaded the cord it might be a sarcoma, though nothing was brought forward to indicate the diagnosis of sarcoma.

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29. Ebinger, F.: Lipom im Rückenmarks-Kanal einer Kuh., Schweiz. Arch. f. Tierh. **43**:177, 1901.

An intramedullary lipoma was found by Kölliker in the lumbar enlargement of the spinal cord in a rabbit. The tumor was situated in the angle formed by the dorsal and ventral horns of the gray matter and in the adjacent white columns.

The third intradural lipoma is that described by Braubach,[30] in 1889, and is an almost exact counterpart of the tumor I am presenting. It is almost identical in position, size, the manner of its growth and in the involvement of the cord. It also had a free pole at both its upper and lower extremities, and it could not be separated from the cord. Both tumors occurred in children, and in both the symptoms appeared early in life. Braubach's patient died at the age of 5. The child had had no illness and was apparently well until the age of 14 months, when suddenly, during the night, it began to have severe cramps in both upper and lower extremities. These attacks were extremely painful and occurred frequently. Some time later the parents noticed that the child was weaker and could not walk as well as before, but would hold on to chairs and tables. Within a few months after the onset the child could no longer walk, and at the end of the second year complete paralysis of both legs and of the right arm was present, with incontinence of the rectum and the bladder and marked contractures. The left arm remained normal. At necropsy a large fusiform intradural lipoma, 12 by 3 by 2 cm., was found beneath the dura, and between the pia and arachnoid (fig. *2A*). The tumor extended from approximately the fourth cervical to the fourth thoracic segments. The outer surface was smooth except in a few isolated places, where it was adherent to the dura. The spinal cord was pushed to the left, and the nerve roots on the right were elongated, stretched and flattened. The tumor appeared to be of the typical fatty form. It could not be freed from the spinal cord and had large connective tissue septums running through it. Microscopically it was a pure lipoma, which it was thought arose from the pia-arachnoid. Except for the fact that the progress in this case was more rapid and the signs more advanced than in mine, the two cases are almost identical.

Spiller, in 1899, found at necropsy a small fusiform lipoma within the filum terminale in a tabetic patient. The pia was infiltrated with fat cells, but none were found exterior to the pial sheath, and the nerve roots were adherent.

Root, in 1907, described a fatty tumor, 1.5 by 5 mm., said to be beneath the dura, in a woman, aged 43, who had had symptoms of spinal cord compression for fifteen years and had been bedridden for a number of years before operation. During the paralytic period she had borne two or three healthy children. Important details concerning the

30. Braubach: Ein fall von Lipombildung der Rückenmarkshaüte, Arch. f. Psychiat. **15**:489, 1889.

tumor, such as its relationship to the cord and the degree of compression of the cord, are not stated.

Wolbach and Millet [12] found an extensive lipoma in a child, aged 10 months, the tumor reaching from the medulla to within 2 cm. of the filum terminale. The cord was markedly compressed and flattened, measuring from 2 to 4 mm., and in places was almost completely replaced

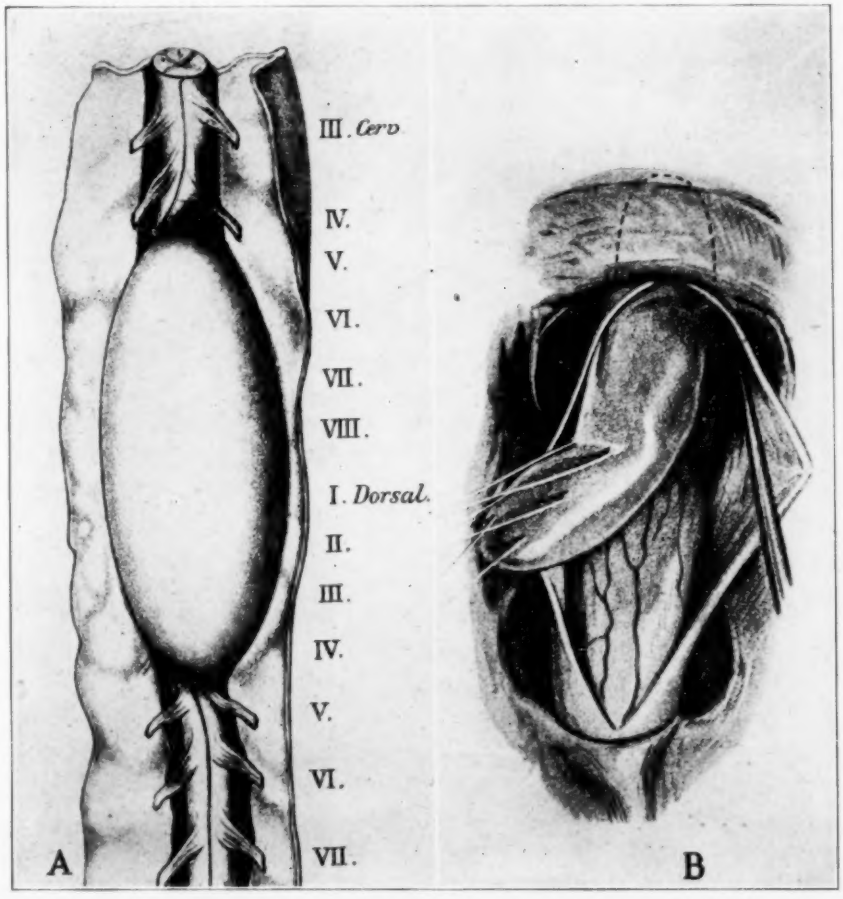

Fig. 2.—*A*, intradural lipoma, extending from the fourth cervical to the fourth thoracic segments, found at necropsy (Braubach: Arch. f. Psychiat. **15**:489, 1889). *B*, intradural lipoma extending from the medulla to the fourth cervical segment (Oppenheim, H., and Borchardt, M.: Deutsche Ztschr. f. Nervenh. **60**:1, 1918).

by fat tissue, particularly in the cervical region (fig. 3). Here the dorsal columns were separated in bundles by connective tissue strands, continuous with the pia, and containing numerous fat cells. There was no evidence of embryologic fat tissue in the tumor. In addition to the

extensive intradural lipoma, cleft palate, hare lip, absence of the right kidney and ureter and internal hydrocephalus were found. The hydrocephalus could possibly be explained by the tumor extending into the medulla and obstructing the free outflow of the cerebrospinal fluid. Wolbach and Millet questioned whether such lipomas ever occurred except in connection with congenital malformations.

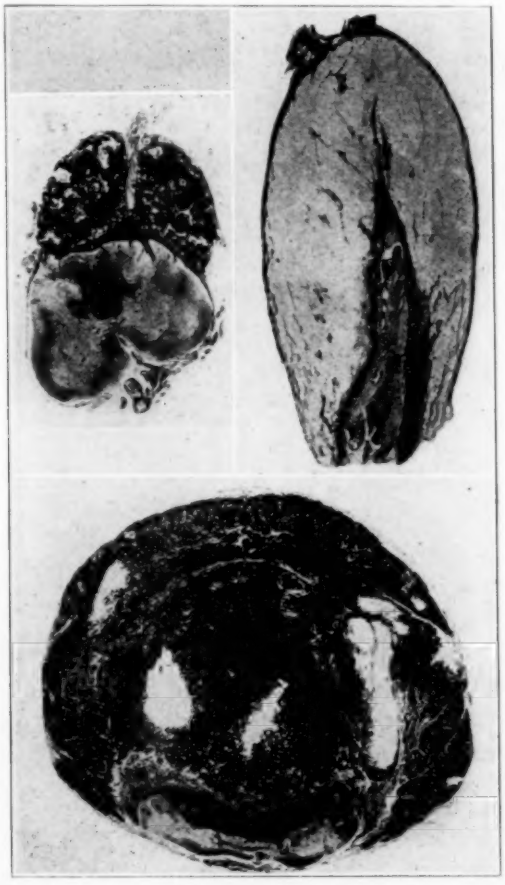

Fig. 3.—Sections from intradural lipoma in an infant, aged 10 months (Wolbach, S. B., and Millet, J. A. P.: Boston M. & S. J. **168**:681, 1913). This tumor extended from the medulla to the end of the spinal cord.

Oppenheim and Borchardt [31] found an intradural lipoma, about 6 by 4 cm. long, extending from the medulla to the fourth or fifth cervical segments, in a physician, aged 44. Thirteen years previously he had

31. Oppenheim, H., and Borchardt, M.: Weitere Beitrag zur Erkennung und Behandlung der Rückenmarks-geschwülste, Deutsche Ztschr. f. Nervenh. **60**:1, 1918.

developed a feeling of weakness in the shoulder region, although three years later he was able to serve as surgeon in the Russo-Japanese war. About 1904-1905, he noticed paresthesias in the fourth finger of the left hand and weakness of the left leg. This was followed, in 1907, by paresthesia of the whole of the left hand, with burning in the left foot and weakness of the left leg. In 1908, paresthesias of the right hand and weakness of the right leg appeared. The weakness gradually progressed until 1910, nine years after the appearance of the first symptom, when the patient became bedridden and tetraparaplegic. The left side showed more marked paralysis than the right. There was some atrophy of the muscles of the neck. Fourteen years after the onset, he was operated on by Borchardt, and an intradural lipoma was found which apparently invaded the cord. The tumor could not be removed, but in spite of this the patient showed some improvement (fig. *2B*).

Thomas and Jumentié,[32] in 1912, found an ovoid intradural lipoma, about 1.5 by 3 cm., which had almost completely replaced the conus. The remaining part of the conus was pushed ventrad and was markedly compressed. The fatty tissue followed the interstitial spaces of the cord, and nerve roots were to be seen running through the tumor.

Ritter [11] found an intradural lipoma extending from the medulla to the first and second thoracic segments, in a man, aged 40. The clinical history of this patient differs from that of other patients with intradural lipomas in that the clinical course lasted only three and a half months. The illness began with pain in the region of the sciatic nerve on the right side; the patient died, with a complete tetraplegia, three and a half months after the onset. Within a few weeks after the onset, the gait had become unsteady; he felt as if he were walking on rubber. At this time, sensation was said to be normal; the deep reflexes were exaggerated. Within eight weeks he was no longer able to walk, and marked weakness in the upper extremities was noted; only with difficulty was he able to lift his arm to a horizontal position. Marked exaggeration of all deep reflexes, incontinence of the bladder and rectum and marked sensory changes were found. The lower extremities were held in spastic extension and could be flexed with difficulty. Necropsy revealed a lipoma extending from the medulla to the second thoracic segment, in places invading the cord, which was pushed to the left. The medulla was flattened, and at the region of the seventh cervical segment the tumor had replaced the spinal cord almost completely. So marked was the infiltration that Ritter felt that operative removal would have been impossible.

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32. Thomas, S., and Jumentié, J.: Lipome de cone terminale, Rev. neurol. **21**:222, 1912.

Extradural lipomas, as has been said, show considerable variation in histologic structure. Only two, with possibly a third, were pure lipomas; the remainder were mixed tumors. Both pure lipomas were found in children; the first was an extremely large tumor extending from the seventh cervical to the second lumbar vertebra, while the other tumor was a circumscribed mass at the first and second thoracic vertebra. Clinical signs were present in the latter case for two years, and in the former for a year and a half, death being due in the first case to convulsions of unknown origin and in the second to scarlet fever. Of the mixed lipomas, two were associated with tumors of the spinal cord itself, one an angiolipoma associated with a glioma and the other an angiolipoma associated with an angioma of the cord, so that in these cases it has been impossible to separate the signs due to intramedullary tumor from those caused by the extradural tumor. The duration of the illness in both cases was relatively short. The circumscribed angiolipoma found by Liebscher showed spinal cord signs for four and a half years. The angiolipoma described by Berenbruch was really a paravertebral tumor which had grown secondarily into the canal. Extradural tumors which show such differences in structure and growth would naturally present dissimilar clinical signs, and in a study of the more common extradural tumors the same variation in symptomatology and clinical course was found.

The intradural lipomas, on the other hand, form a most interesting group of spinal cord tumors. They are interesting because of their appearance in early life, the extreme size that they may attain, and the apparent invasive power of the tumor. They are furthermore unusual in that they may present signs for a number of years—ten, twelve and fifteen.

The intradural lipomas are found both in the spinal cord and in the region of the conus and cauda equina. The latter group should be considered separately. They comprise three tumors, two of which were found coincidently at necropsy in tabetic patients, with no clinical signs due to the tumor. In the third case signs referable to the conus were present, and from the meager report given the signs correspond to the syndrome usually associated with conus lesions. However, the interesting point is that the neoplasm, though a pure lipoma, included the conus and in part almost replaced the cord tissue, so that operative removal would not have been possible.

The remaining intradural lipomas which compressed the spinal cord may be divided into two groups: those found in children, and those in adults.

The three lipomas found in children gave signs at about the first year. One child died at the age of 10 months, and in the two remaining instances—Braubach's and my case—signs referable to the tumor were present in the second year. In both, the tumor extended from approxi-

mately the fourth cervical to the fourth thoracic segments. In both, the tumor had two free poles and involved the spinal cord, making operative removal impossible. In Braubach's case the tumor was found at necropsy, and no attempt to operate had been made. In the case of Wolbach and Millet the tumor was nearly as long as the spinal cord, and also involved the cord. Thus, in all three cases the tumors were extensive, appeared early in life, were mainly in the region of the cervical enlargement, and involved the cord in such a manner as to make removal impossible.

Of the tumors in adults, two were in the cervical region and the remaining two in the midthoracic region. One of the latter was an intramedullary tumor without extramedullary extension. This tumor had existed for twelve years with gradually increasing paralysis of both legs. Such long duration, even when the tumor involves the cord, seems to be characteristic of intradural lipomas. Apparently, fat tissue is well borne in the cord tissues, since only after a number of years is spinal cord function destroyed. In my case the tumor had presented some signs for approximately ten years, and yet the patient was able to walk, though with spastic gait. In Turner's case signs were present for twelve years, in Oppenheim and Borchardt's case for thirteen years, and in Root's case for fifteen years. This long duration might be explained by the fact that pure lipomas are soft and cause relatively little pressure.

On the other hand, the tumor may take on rapid growth, as in Ritter's patient, who died within three and a half months of the onset of the symptoms. The early death in Wolbach and Millet's case was probably due as much to the extent of the tumor as to the rapidity of growth. Wolbach and Millet felt that there was no histologic evidence that they were dealing with a rapidly growing tumor of embryologic fat cells.

The "invasive" power of the tumor is extremely interesting. All of the cases which presented an extramedullary tumor showed an intramedullary extension, with the exception of Root's case. The description of the latter is meager and wanting in many details. In Turner's case the tumor was completely intramedullary, without any extramedullary extension. Borchardt was unable to separate the tumor from the spinal cord, but found that the tumor extended into the cord, which forced him to refrain from further operative procedure. Ritter found that the tumor replaced the spinal cord, especially in the region of the seventh cervical segment. In my case the tumor could not be separated from the cord except at the two poles, and the tumor extended directly into the cord. It was impossible to determine from the tissue whether the tumor arose primarily in the pia and secondarily invaded the cord, or whether the tumor was primary in the cord and secondarily extended as an extramedullary growth.

OPERABILITY

Removal of intradural lipomas has not been possible in those cases in which operation was performed, nor would it have been possible in those found postmortem. Hence, it would probably be well not to persist too tenaciously in attempts to separate such tumors from the spinal cord, bearing in mind that those so far reported have been both intramedullary and extramedullary, without any line of demarcation within the cord.

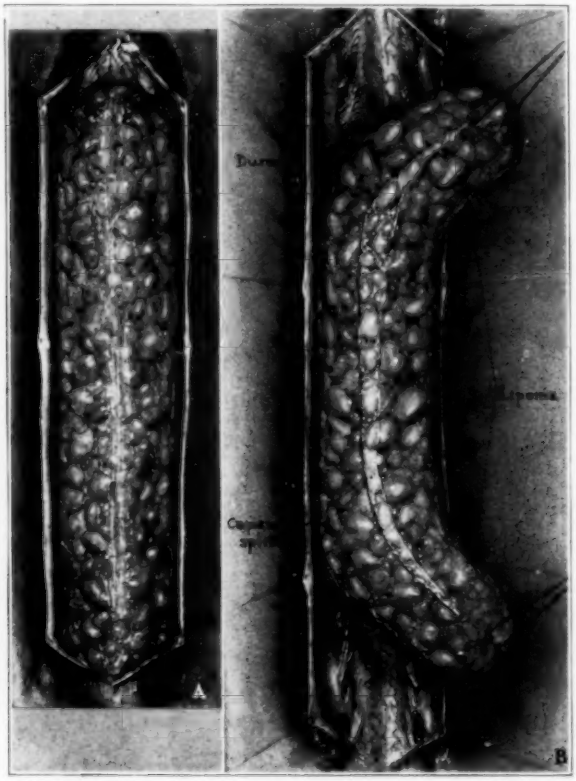


Fig. 4.—*A*, exposure of cervical and upper thoracic cord. The dura is open and an intradural lipoma is seen lying beneath the pia-arachnoid; *B*, both poles of the lipoma have been pulled outward. When it was found that the tumor could not be removed (except the free poles) the capsule of the tumor was split and the dura left open.

In my case the nerve roots ran through the tumor, and all attempts to separate it from the spinal cord were futile. The operation of choice would seem to be to split the capsule in the midline and to leave the dura open. The essential point is to split the pia-arachnoid, since this forms a capsule and prevents free extrusion of the tumor if allowed to enclose

it. By splitting the capsule and leaving the dura open under a wide decompressive laminectomy, pressure on the cord would be relieved as much as possible.

DIAGNOSIS

Spinal cord neoplasms in children, not including sacral tumors or extensions from contiguous structures, are extremely rare. Stursburg, Antoni and Frazier's collections from the literature brought forth only one instance of spinal cord tumor in children less than 10 years of age. The records of the Neurological Institute show only two instances of spinal cord neoplasm in children under 10 years of age. Only one instance of spinal cord neoplasm in children between the ages of 10 and 15 is to be found in the Neurological Institute records, other than the case here reported, and in no instance was the tumor intradural. Nevertheless, in spite of the rarity of spinal cord tumors in children, an intradural lipoma is to be considered in any child—no matter how young—who shows signs of spinal cord compression, especially if other fatty tumors or congenital defects are present. Lipomas especially should be considered when spinal cord compression signs have existed for a relatively long time. The long duration of symptoms would point rather to intradural lipoma than to an extradural tumor. Of the two cases of spinal cord tumors in children under 15, the average duration was a matter of months, while the duration of intradural lipomas is in terms of years. The long duration of signs of spinal cord compression in adults, as well as in children, should make one consider the possibility of an intradural lipoma in the differential diagnosis. The neurologic signs caused by an intradural lipoma are no different from those in any other spinal cord tumor, and on the neurologic signs alone I do not believe that a differentiation can be made.

The presence of a tumor of considerable size within the vertebral canal in a young person might conceivably cause enlargement of the canal. Constant pressure exerted in the young on pliable bone might well show its effect in producing a larger vertebral canal than is usually found. Such an enlargement seen in a roentgenogram would of course be a valuable sign, not only as to the presence of a tumor, but also to its exact localization. In this patient, the vertebral column at operation was found considerably enlarged, the laminae being almost as thin as paper. In reviewing the roentgenograms of this patient taken over a number of years, it is apparent that an enlargement of the vertebral canal existed even five years before operation.

The presence of pressure atrophy of the arches and laminae of the vertebrae in spinal cord neoplasm has been frequently observed at operation, not only in children but also in adults, usually in association with large extradural tumors. It is remarkable that pressure atrophy

of the bone is possible even in older persons without an associated, almost complete destruction of the extremely soft spinal cord. As pointed out in a previous study of extradural tumors, pressure atrophy of bone, with relatively slight compression of the spinal cord, was considered due, possibly, to the noncompressible fluid buffer which surrounds the cord. This buffer so distributes the pressure that extradural tumors press directly on the bone but only indirectly on the spinal cord, through the fluid buffer. Hence, atrophy of the bone occurs earlier than compression of the spinal cord.

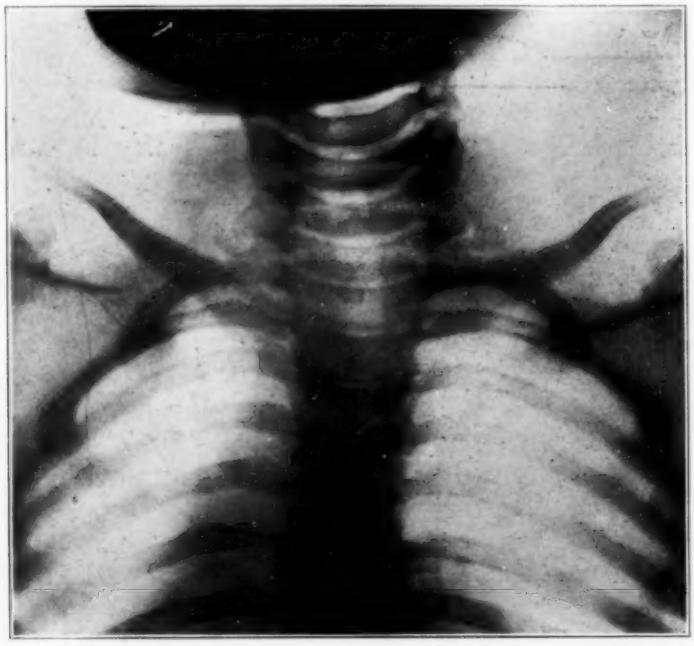


Fig. 5.—Enlargement of the cervical vertebrae and the vertebral canal due to the presence of an intradural lipoma.

REPORT OF CASE

Clinical History.—W. L., a first child, was born normally, no instruments being used; he was not blue and there was no trauma; he weighed about 10 pounds (4.5 Kg.) at birth and was always fat. He was breast fed for eighteen months and then fed by bottle. He began to sit up at 7 months and to talk at 9 months. He did not crawl and did not begin to walk until the age of 3 years. The gait then was unusual; the feet were held far apart, and the mother noticed that something was wrong. When he was about 2, a small superficial lump, the size of an egg and freely movable, was noticed in the back of the neck. The child was taken to a physician who advised removal of the tumor, but operation was refused. At the age of 3½, the boy was brought to the United States and taken to the New York Orthopedic Hospital, in May, 1919, with the complaint of weakness of

the legs since 1915. The boy was well developed and well nourished; the height was 44 inches (112 cm.). There was increase in the patellar reflexes and slight spasticity of the adductors. A bilateral Babinski sign was present. Because of contracture of the Achilles tendon, and genu recurvatum, a splint was applied, and the right Achilles tendon cut. A brace was worn for a year and a half. In July, 1921, the condition was essentially unchanged, although the mother thought that the boy walked better; the base was not so wide, and the feet no longer dragged. At this time there were pain and slight stiffness of the neck, and a posterior bulging was noticed in the upper thoracic region. Roentgenograms of the cervical spine showed nothing of importance. In August, 1921, the spasm of the neck had disappeared. The patient was observed irregularly by visiting nurses and also at the dispensary until December, 1921. During this period he was receiving gymnastic therapy and showed some improvement. He had occasional pains in the arms and legs. Tonsillectomy was performed. The Pirquet and Wassermann tests were negative.

In October, 1922, a lipoma was removed from the back of the neck at the Skin and Cancer Hospital. Roentgenograms at this time revealed bilateral cervical ribs with thinning of the disks between the third, fourth and fifth thoracic vertebrae. The Wassermann and Pirquet tests again were negative. On account of the pain in the back of the neck, in the lumbar region and in the right arm, the patient was readmitted to the Orthopedic Hospital in November, 1922. At this time he had a general adenopathy, spasm with hyperextension of the upper thoracic spine and slight limitation of head flexion. All the muscles were slightly spastic with slight adductor spasm. There was no spinal tenderness. Spinal fusion of the first to the eighth dorsal vertebrae was performed in November, 1922, and a diagnosis of Pott's disease was made, although the roentgenograms showed nothing new. The patient was discharged from the country branch in March, 1925. During his stay in the country, notes were made of both the presence and the absence of a bilateral Babinski sign and clonus. The spasticity had extended to the right arm, but otherwise the neurologic examination was said to give negative results. A Pirquet test again was negative to both human and bovine strains.

In April, 1925, atrophy and weakness of the right biceps, triceps, forearm and intrinsic muscles of the hand were noted. A paradoxic right triceps reflex was elicited on tapping the biceps tendon. There was some spasm and cramp in the right little finger. Examination still showed no active tuberculous lesion.

Because of the recurrence of severe pain in the neck and rather prominent neurologic signs, the patient was again admitted to the Orthopedic Hospital, Oct. 7, 1925, and was referred to the Neurological Institute by Dr. Alan Smith and Dr. Henry Riley.

Examination.—The patient was well developed and nourished and was not acutely ill. He held the right hand rigid and slightly to the left. There was slight genu valgum, and the knees were partly flexed. The gait was unsteady, and somewhat staggering, with the knees held partly flexed; he walked with a widened base and scraped the toes on the floor. The gait may be characterized as spastic-ataxic. The patient was able to stand on his toes but not on his heels. There was increase of the associated movements of the trunk, arm and head; the increase in the upper extremities was more marked on the right than on the left, the right arm being abducted and swung rather freely, with the wrist held in extension. Standing with the feet together and the eyes open, the patient swayed; this was accentuated when the eyes were closed. He stood very poorly on either foot, with the eyes open or closed. The finger-to-nose and finger-to-finger tests were well performed on each side. The finger-to-thumb test was performed normally with

the left but very poorly with the right hand. The toe-to-object above the recumbent patient test showed definite wavering in each leg. The heel-to-knee and along-shin tests showed marked ataxia in each leg. Succession movements showed marked disturbance in the right arm.

Skilled Acts: The patient was unable to use the right hand for skilled acts. The handwriting was poor.

The Deep and Superficial Reflexes.—These are shown in table 1.

Muscle Strength: In the right upper extremity, marked flaccidity and weakness were present in the trapezius, deltoid, triceps, and flexors and extensors of the fingers. The left upper extremity showed definite but less marked weakness. Marked weakness was noted in adduction of the upper extremities on each side, the right side more than the left. There was some general weakness in the muscle groups of the lower extremities.

Muscle Status: Atrophy was observed in the right thenar and hypothenar eminences.

Vertebral Morphology: The patient had a rigid thoracic spine. There was exquisite tenderness over an area corresponding to the third thoracic vertebra.

TABLE 1.—*Deep and Superficial Reflexes* *

Deep	Left	Right	Superficial	Left	Right
Jaw	2	2	Ciliospinal	Present	Present
Pectoral	1	2	Upper lateral abdominal	0	0
Biceps	1	0	Lower lateral abdominal	0	0
Radial	0	1	Cremasteric	0	0
Triceps	1	1†	Plantar flexion	0	0
Ulnar	0	0	Babinski sign	Present	Present
Wrist	0	0	Chaddock sign	Doubtful	Present
Suprapatellar	4	2+	Oppenheim sign	0	0
Patellar	3+	3	Gordon sign	0	0
Hamstring	3	3			
Achilles	5	4			
Periosteal, crossed	0	0			
Periosteal, uncrossed	0	2			

* In this table, 0 indicates absent; 1, slight; 2, active; 3, very active; 4, transient clonus (on percussion); 5, with permanent clonus.

† With a spread to the deltoid and on one occasion to the biceps.

There was no tenderness anywhere else. The head was held fairly rigid and could not be flexed; slight passive extension and lateral movements were possible.

Sensory Examination: The results are shown graphically in figure 6. Vibratory sensibility was absent over the tibias and diminished over the anterior superior spines of the ilia. It was felt normally over the sternum and the vertebral spines. Muscle tendon sensibility showed definite defect in the toes of each foot.

Cranial Nerves: The ocular fields and fundi were normal. The vision was 20/20 in each eye. The right pupil was slightly smaller than the left. The pupils were round, central in position and reacted briskly to light, both direct and consensually, and in convergence and accommodation. There was no diplopia and no exophthalmos, but there was slight exophthalmos of the right eye. The movements of the eyes were normal. There was no nystagmus in any direction, although on one occasion a slight nystagmus in all directions had been reported. The left palpebral fissure was slightly wider than the right.

Systemic Examination: The hair, teeth and nails were normal. The skin showed a scar extending from the first to the eighth thoracic vertebra in the midline dorsally. On the day preceding the examination the right ear had been constantly red while the left had been normal in color. The thyroid gland, gonads

and tonsils appeared normal. There was slight enlargement of the cervical nodes; no epitrochlear or inguinal glands were palpable.

Mental Status: This was entirely normal.

Laboratory Examinations: The blood showed: hemoglobin, 75 per cent; color index, 0.95; red blood cells, 3,900,000 per cubic millimeter; white blood cells, 10,000 per cubic millimeter; neutrophils, 72; small lymphocytes, 26; large mononuclears, 26, and eosinophils, 2 per cent; slight anisocytosis; urea, 26.1 mg., urea nitrogen, 12.2 mg., and sugar, 0.1 Gm. per hundred cubic centimeters. The spinal fluid appeared pink at the time of withdrawal but on examination in the laboratory was yellowish, with fibrin; red blood cells were present; globulin was 4 plus; the Wassermann test was negative. A manometric study revealed a practically complete block (fig. 7). The urine was a light amber; examination showed: acid; specific gravity, 1.028; no albumin or sugar; indican, increased; a few leukocytes, and calcium oxalate crystals were present.

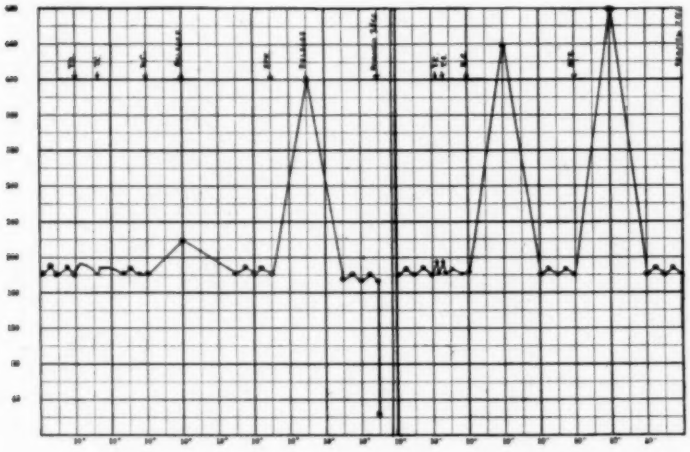
Fig. 6.—Sensory changes to touch and pain and temperature. Touch everywhere interpreted correctly, but slight and definite inequality. Right lower extremity and torso "more ticklish." The cross hatching indicates slight diminution.

Diagnosis.—Before operation the diagnosis was intramedullary tumor of the midcervical region. The postoperative diagnosis was: lipoma, intramedullary and extramedullary, apparently arising out of the cord or secondarily invading the cord; no line of demarcation.

Operation.—First Stage: Nov. 11, 1925, the fifth, sixth and seventh cervical laminae were removed and subsequently also the fourth, third and second cervical laminae. The vertebral canal in the cervical regin was approximately twice as large as is usually found. The arches were thin—almost the thickness of paper—showing marked pressure atrophy. The inner table was smooth. The dura completely filled the vertebral canal and showed no pulsation. A mass could be felt beneath the dura. The pia-arachnoid could not be identified, but on exposing the dura a fatty tumor mass presented; it was well encapsulated and extended laterally as far as could be seen. It was thought that the pia-arachnoid formed the capsule of the tumor.

The lower pole of the tumor extended below the operative field; the upper pole extended as high as the second cervical segment. The tumor showed a definite capsule, had a typical fat appearance and was nonvascular. Its lateral limits could not be made out, even though bone was removed laterally as far as possible on both sides and the dura on the right side was split to give the widest possible exposure. An attempt was made to expose the upper pole. When the upper pole was reached, the fourth and third cervical nerve roots were seen in the capsule of the tumor. The right fifth, sixth and seventh roots were seen coming out of the tumor laterally.

A certain number of roots on the left side also were seen to come out of the side of the tumor. An attempt was made to find a line of demarcation at the junction of the upper pole of the tumor and the cord, but this could not be done. There seemed to be no line of separation. The tumor apparently surrounded or arose out of the cord and could not be separated from it. Small parts of the tumor were removed, but the major portion was left. The patient's condition



A B

Fig. 7.—*A*, manometric study of the cerebrospinal fluid. *B*, normal chart, showing the typical rise on touch compression, deep compression, straining and removal of fluid. *T. C.* indicates touch compression; *D. C.*, deep compression; *Str.*, straining. The marked fall in level of the fluid after removal of 7.5 cc. in *A* as compared with fall in pressure shown in *B* should be noted.

became poor; the pulse rose to 160 and the blood pressure dropped. It was therefore deemed inadvisable to continue. The dura was left open except over the second and third cervical segments, where it was closed.

Second Stage: Jan. 30, 1926, the incision was carried down through the old scar of the spinal fusion and also through the scar of the previous laminectomy. The first and second thoracic vertebrae were exposed, and the laminae removed to bring the dura into view. The incision was then carried up through the former opening as far as the occiput, exposing the previous laminectomy. When the dura was freed above the second thoracic vertebra, a mass of fat protruded and hung back over the dura. This was the lipoma which had protruded from the previous operation, since the dura at that time was left open. The fatty tumor was followed upward to the region of the second cervical vertebra. It bulged markedly. At the upper limit cerebrospinal fluid escaped in a large gush. Imme-

diately, complete collapse occurred, so that the lipoma went within or at least to the level of the dura and no longer protruded. The lipoma as now exposed extended from the second cervical to the second thoracic vertebra and beyond. Attempts were made beneath the second thoracic vertebra to find the line between the cord and the lipoma by curetting through the lipoma, but even when this had been carried to a considerable depth no line of cleavage was found. It was deemed inadvisable to proceed further by this method. An attempt was then made to expose further to see if the lower limit could be found and the third and fourth thoracic laminae were removed. The distal pole of the tumor was found beneath

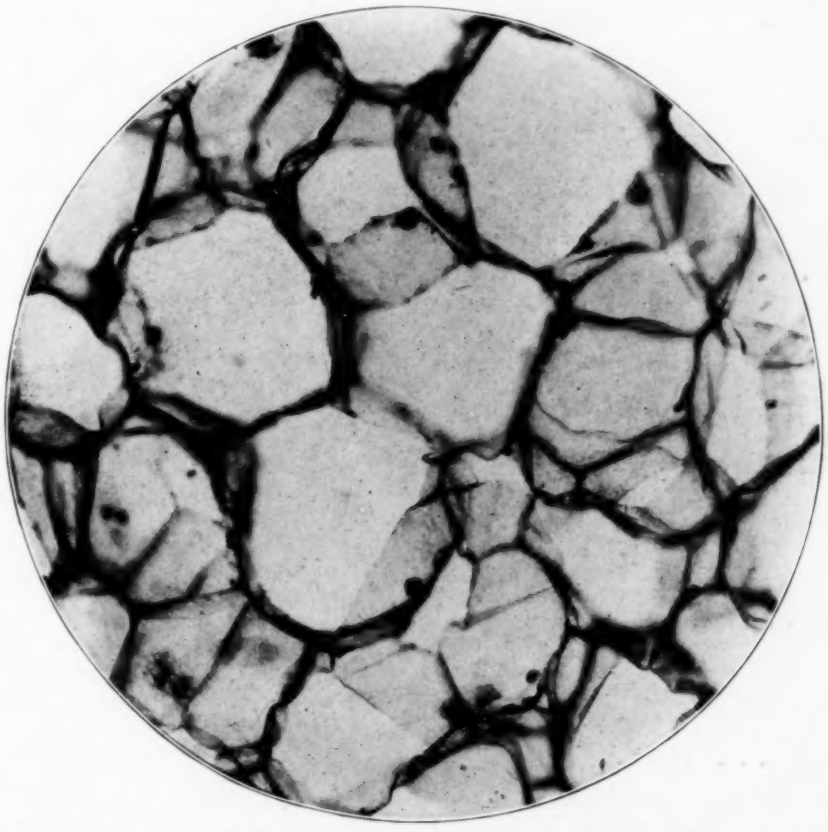


Fig. 8.—Cross-section of intradural spinal lipoma showing typical fat cells. Hortega silver carbonate stain showing cell outline.

the fourth thoracic vertebra. This part of the tumor was freed from the cord, no definite line of cleavage being found. Three dorsal roots were torn as the most caudal part of the tumor, measuring approximately 2 or 3 cm. or possibly more in length, was removed. This could not be separated farther than approximately opposite the first or second thoracic vertebrae. Here, as at the cephalic end, the tumor entered the cord as if it had either grown out of the cord or had invaded it secondarily. The remaining tumor capsule was then split and the dura left open.

Course.—Following the operation the patient showed marked improvement in gait and ability to get around. The sensory changes disappeared, and on discharge

no changes could be detected. The ankle clonus disappeared, and the knee reflexes have become practically normal. There is, however, considerable disability in the use of the right hand, due probably to injury to the seventh cervical root.

TABLE 2.—*Intradural Spinal Lipomas*

Author	Year	Type	Age	Cord Invaded	Location	Duration	Operation or Necropsy
1. Gowers.....	1876	Myelipoma	..... (Tabetic)	Not invaded	Conus	....	Necropsy
2. Turner.....	1888	Lipoma	54 years, 6 months	Intramedullary	Midthoracic	12 years	Necropsy
3. Braubach....	1889	Lipoma	5 years, 6 months	Cord invaded	Fourth cervical to fourth thoracic	3.8 years	Necropsy
4. Spiller.....	1899	Lipoma	..... (Tabetic)	Within filum terminale	Filum terminale	....	Necropsy
5. Root.....	1907	Lipoma	43 years	Not invaded	Midthoracic	15 years	Operation (improved)
6. Wolbach and Millet	1913	Lipoma	10 months	Cord invaded	Second cervical to first sacral	Congenital	Necropsy
7. Oppenheim and Borchardt	1918	Lipoma	44 years	Cord invaded	Medulla to cervical	13 years, after 9 years bedridden	Operation (improved
8. Thomas and Jumentié	1912	Lipoma	.....	Cord invaded	Conus	....	Necropsy
9. Ritter.....	1920	Lipoma	40 years	Cord invaded	Medulla	3½ months	Necropsy
10. Stookey.....	1926	Lipoma	11 years	Cord invaded	Fourth cervical to fourth thoracic	11 years	Operation (improved)

TABLE 3.—*Extradural Spinal Lipomas*

Author	Year	Tumor	Age	Duration	Level	Operation or Necropsy
1. Chapelle....	1847	Lipoma	4 years	1½ months	Fourth cervical to second lumbar	Necropsy
2. Albers.....	1842	Lipoma, multiple	....	.....	.....	Necropsy
3. Obré.....	1852	Lipoma	3 years	2 years	First to second thoracic, circumscribed	Necropsy
4. Virchow....	1857	Myxoides lipomatodes, multiple	53 years	11 months	Lumbar and first sacral (?)	Necropsy
5. Hoffmann..	1869	Lipoma, multiple with glioma of cord	43 years	9 months	Lumbar and sacral	Necropsy
6. Berenbruch	1890	Angiolipoma, multiple with angioma of cord	16 years	Few months	Thoracic	Necropsy
7. Starr.....	1895	Lipoma (?)	50 years	4 months	Tenth to eleventh thoracic	Operation
8. Strümpell..	1897	Lipoma	....	.....	.....	Necropsy
9. Liebscher...	1901	Angiolipoma	56 years	4½ years	Sixth to eighth thoracic	Necropsy

TABLE 4.—*Intradural Spinal Lipomas According to Age*

Author	Year	Age	Age at Onset	Duration	Level	Operation or Necropsy
			Children			
1. Braubach.....	1899	5 years	14 months	3½ years	Fourth cervical to fourth thoracic	Necropsy
2. Wolbach and Millet	1913	10 months	Birth	10 months	Second cervical to first sacral	Necropsy
3. Stookey.....	1926	12 years	14 months	11 years	Third cervical to fourth thoracic	Operation
Average.....		6 years		5 years		
			Spinal Cord Adults			
4. Turner.....	1888	54 years	42 years	12 years	Midthoracic	Necropsy
5. Root.....	1907	43 years	28 years	15 years	Midthoracic	Operation
6. Borchardt and Oppenheim	1918	44 years	31 years	13 years	Medulla to fourth cervical	Operation
7. Ritter.....	1920	40 years	40 years	3½ months	Second to fourth cervical	Necropsy
Average.....		45 years		10 years		
			Conus and Cauda Adults			
8. Gowers.....	1876	Tabetic	.....	.....	Conus	Coincident necropsy
9. Spiller.....	1899	Tabetic	.....	.....	Filum terminale	Necropsy
10. Thomas and Jumentié	1912	.....	.....	.....	Conus	Necropsy

CONCLUSIONS

1. Intradural spinal lipomas are heterotopic neoplasms, probably arising from inclusions at the time of the formation of the neural tube. They are usually pure lipomas.

2. Extradural spinal lipomas are usually mixed neoplasms. They may be found associated with intradural neoplasms such as angiomas or gliomas.

3. Intradural spinal lipomas are found more often in those regions of the neural tube which are late to close; namely, the cervical and sacral hiatuses.

4. An intradural spinal lipoma may be a congenital neoplasm giving rise to spinal cord signs first noticeable when a child begins to walk. Spinal cord neoplasm should be suspected in a child with tetraplegia, and a manometric study of the cerebrospinal fluid should be made.

5. Spinal cord signs may at first be indefinite. They are slowly progressive.

6. The duration of symptoms may be for years—the average duration in children was five and in adults ten years.

7. A roentgenogram may show enlargement of the vertebrae overlying the neoplasm.

8. Most intradural lipomas are both intramedullary and extramedullary, without any sharp line of demarcation separating the tumor from the spinal cord. When a line of demarcation cannot be readily

made out, too strenuous attempts to remove the tumor should not be made, since removal of the intramedullary part does not seem to be possible.

9. When not removable, the capsule, usually consisting of the pia-arachnoid, should be split and the dura left open.

DISCUSSION

Dr. Beverly R. Tucker, Richmond, Va.: Is it possible for these lipomas to be secondary to a fat embolus?

Dr. E. Sachs, St. Louis: Last year before the American Orthopedic Association I reported a case of an extradural lipoma and also showed a necropsy specimen of another case that I had obtained in Berlin, in 1911, and expressed the opinion that these extradural collections of fat are not true tumors.

In the literature, I found that in a certain number of these cases (and my case was similar) there was an enlargement of the canal, and though the cord was compressed it was compressed over a bony kyphotic process. The question naturally came up whether this could be an old tuberculosis, a deformity of the spine, the cause of which was unknown, or merely an attempt to fill up the dead space with fat tissue.

Of course that explanation in no way applies in the cases of intradural lipoma. In an almost complete review of the literature beginning with about 1839, I found that practically all the cases of so-called extradural lipomas were not lipomas but were connective tissue tumors.

Since reporting that case, Dr. McKenzie of Toronto told me that he has had a case similar to mine. I presented it before the orthopedists because I believe these patients are probably more frequently seen by the orthopedists, who have considered them tuberculous, with compression. There was clearly no evidence of Pott's disease in my case. Removing the fat and splitting the dura rapidly relieved the paralysis, which had been present for a year prior to my seeing the patient.

Dr. William G. Spiller, Philadelphia: During a considerable number of years, I have seen only two intradural lipomas; Dr. Stookey referred to one of these. The other was a small tumor in the region of the pituitary body. The case to which Dr. Stookey referred occurred at the Salpêtrière. The tumor was found unexpectedly and produced no symptoms. It was small but was a true lipoma. I reported it after it was given to me by Professor Dejerine. It was located in the filum terminale. A tumor such as Dr. Stookey has reported seems to me an extraordinary contribution. Such cases must be exceedingly rare.

Dr. Byron Stookey: I am unable to reply to Dr. Tucker's question as to the possible source of the tumor in a fat-embolus. On general principles, I should think it extremely unlikely, since I do not believe that fatty tumors grow as implantation tumors, which such a source would imply. However, I should prefer to leave this issue to those with more competent pathologic knowledge.

With reference to Dr. Sachs' comment concerning the extradural tumors, I did not present the details of the extradural tumors, which will be included in the printed paper, because I felt that there was no real problem as to their source. Normally, there is a large extradural fatty pad which intervenes between the dura and the vertebrae; hence, it is rather curious that this fatty pad does not give rise to extradural tumors more frequently than it apparently does.

Some of the earlier cases were excluded because their descriptions were too meager to permit one to determine the exact nature of the growth. The first authentic instance of an extradural tumor that I could find was that of Albers

and Chapelle, in 1847. From a study of the published records, I conclude that there can be little doubt that these are real neoplasms and not merely fatty collections as Dr. Sachs implies. In none of the cases here reported was there any important pathology of the bone such as Dr. Sachs has reported. If Dr. Sachs will give me the citation for 1839, I shall be glad to have it. Dr. Spiller made a thorough review of the literature at the time of his report, and if I recall correctly his was the first report of an extradural lipoma.

The extradural tumors which have been reported were in the main mixed tumors. In some instances mixed lipomatous tumors were found, in association with pure lipomas and pure angiomas. One patient had multiple tumors with an angioma as an extremely large tumor; other tumors were pure lipomas, and others pure angiomas. At necropsy, a whole series of fatty tumors from various parts of the body, most of them mixed tumors, all having some angiomatous and some lipomatous tissue, were found. Consequently, from the reports in the literature, I do not see how one could question the existence of the extradural group of fatty tumors. The description given admits of no other conclusion.

Dr. Sachs' opinion that possibly these tumors are merely connective tissue growths is interesting. Of course a fatty tumor is essentially a connective tissue growth with fat. Many lipomas, but not those included in this series, may be found as replacement tumors, such as are seen in spina bifida, called sacral tumors, which are familiar. As has been said, however, these sacral tumors have not been included in this discussion.

ACUTE (EPIDEMIC?) ENCEPHALITIS

REPORT OF A CASE IN A NEW-BORN TWIN WITH HISTOLOGIC OBSERVATIONS *

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Little is known of acute cerebral conditions in early infancy. Yet they may be of great importance in affording an explanation for the development of some of the epilepsies, defect conditions, etc., that prove so difficult of comprehension late in life. For this reason, the observations in the case to be reported, the opportunity to study which I owe to Dr. Kantor, are deemed worthy of record. The case is of especial interest by reason of the fact that, clinically, nothing was observed that suggested the presence of an acute infection of the brain. Unfortunately, this had the advantage of rendering the clinical study incomplete, and the history is, in consequence, somewhat meager.

REPORT OF CASE

History.—The child, a twin, was born, Jan. 22, 1926, at the North Chicago Hospital by a normal delivery; it weighed 6⅜ pounds (2.9 Kg.), had club feet, hypospadias, an imperforate anus and congenital cystic kidneys; the ulna and the thumb on the right were missing. The child died on the fifteenth day after birth. For two days preceding death, the child had "a temperature." The symptoms evidently were not sufficiently striking to call for detailed clinical study.

Pathologic Observations.—The brain and upper portion of the cervical cord were delivered to my laboratory in a 10 per cent solution of formaldehyde. The meninges were slightly opaque and thickened, but the general external appearance of the brain was normal. Frontal sections revealed macroscopic hemorrhages in the basal portion of the pons and the right lenticular nucleus; the ventricles were not dilated.

Microscopic Examination.—The most striking changes were marked perivascular infiltrations (fig. 1), which were especially evident in the pons—the basal as well as its tegmental portion—and much less so in the cortex. The infiltrating cells were mainly of polymorphonuclear type (fig. 2) especially in the basal portion of the pons; lymphocytes also were numerous, while plasma cells were rare. The infiltrating cells were usually confined to the adventitial spaces of Virchow-Robin which were greatly distended. In some instances, the rows of infiltrating cells were so dense that the lumen, especially of the smaller blood vessels, was markedly narrowed. The contents of the Virchow-Robin spaces often transgressed the limits of the spaces and invaded the surrounding parenchyma (fig. 2). Here they often gathered in large masses,

* From the pathology laboratories of the Research and Educational Hospitals, University of Illinois and the Illinois State Psychopathic Institute.

forming nodules (fig. 3) that occasionally were near blood vessels, but often without any connection with them. In many nodules the predominant cells were not polymorphonuclear but rod or gitter cells, or lymphocytes, frequently mixed with blood pigment. In some blood vessels the perivascular spaces also showed a great number of gitter cells filled with lipoids, and, in addition, minute granules. These were densely stained, loosely attached to one another and often formed circles. They were of frequent occurrence also in the parenchyma (fig. 4) and the choroid plexus (fig. 5). Generally they were especially in evidence in areas in which the polymorphonuclear elements were common.

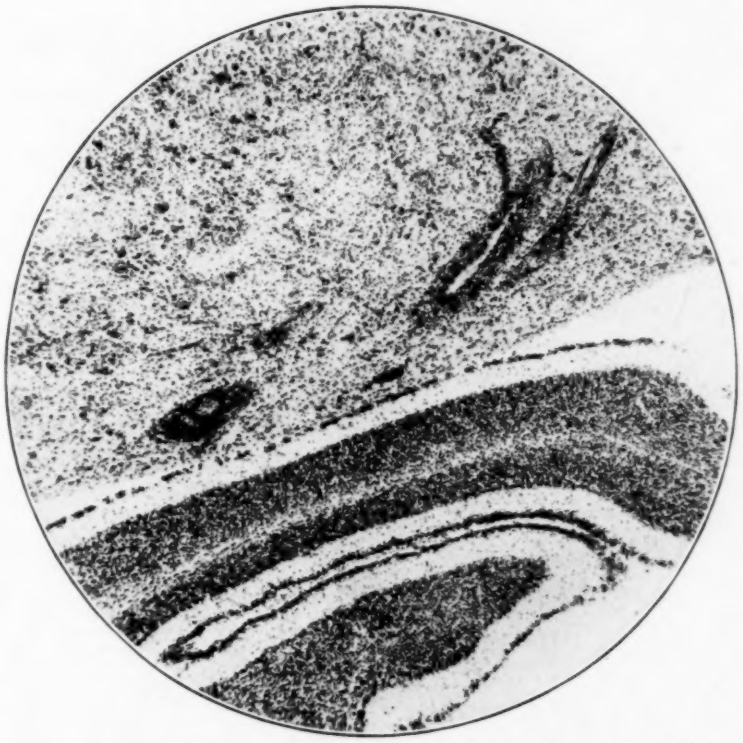

Fig. 1.—Pons and cerebellum. Several blood vessels in the pons, above, are infiltrated; below is the cerebellum. The black bodies in the upper part of the picture are glia cells filled with blood pigment. Toluidin blue, × 42.

The blood vessels, aside from the dense adventitial infiltrations, generally showed progressive phenomena. Their endothelial and adventitial cells were unusually well developed; their nuclei were rich in chromatin and were hypertrophied, especially in the capillaries, many of which were empty and exhibited signs of budding or new capillary formation. Perivascular or adventitial infiltrations, which were so marked around the arterioles and venules, were rare around the capillaries.

Some areas in the pons and in the right lenticular nucleus exhibited hemorrhagic foci which were scattered among the blood vessels and generally were quite numerous. The foci consisted of dense masses of red cells and fibrin

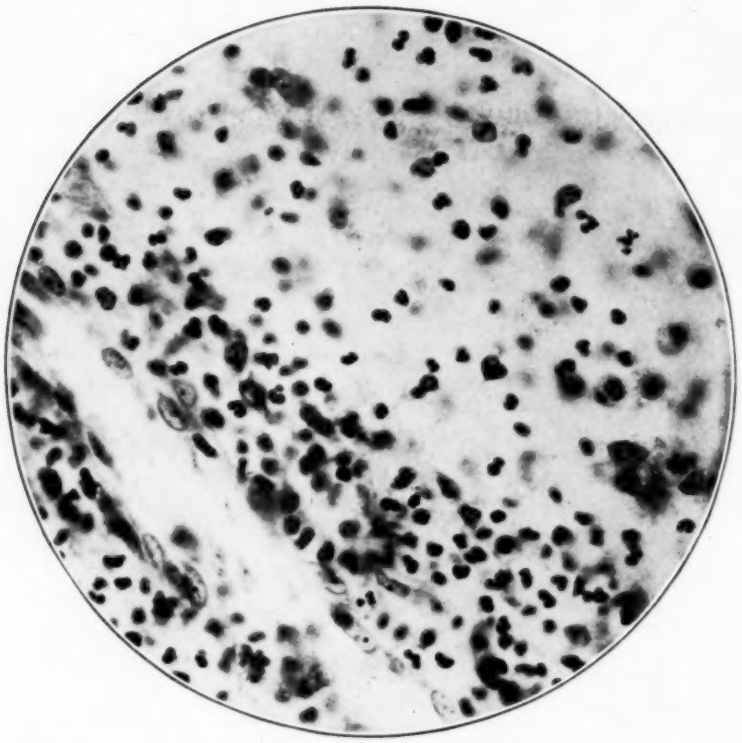

Fig. 2.—Perivascular infiltrations (to the left) under a higher magnification. Polymorphonuclear leukocytes predominate and also invade the parenchyma (to the right). Toluidin blue, × 440.

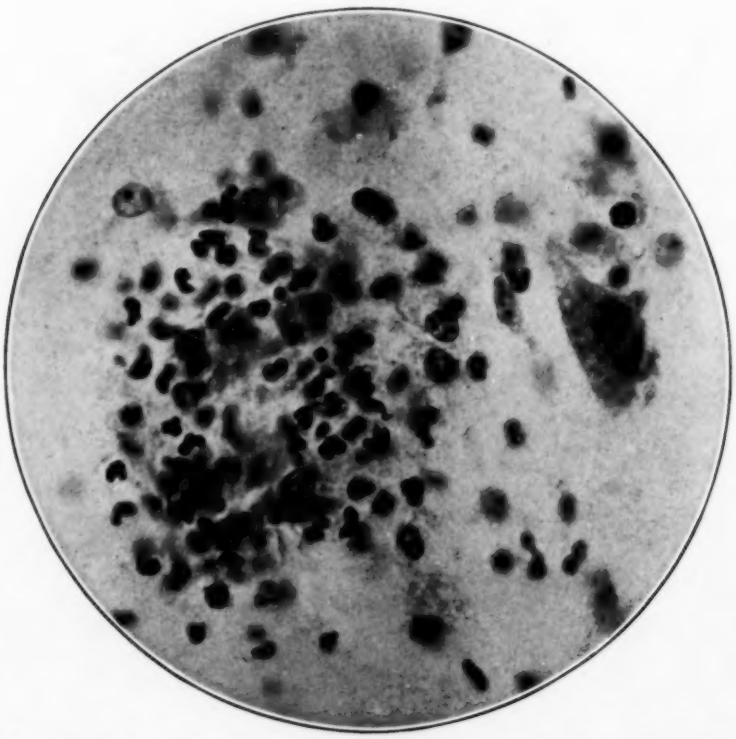

Fig. 3.—Nodule made up chiefly of polymorphonuclear leukocytes. To the right is a changed ganglion cell. Toluidin blue, × 560.

fibers, were irregular in shape and did not show any relationship to the blood vessels or capillaries. They infiltrated the parenchyma, over which were scattered numerous glia cells filled with blood pigment. Such glia cells also were present in areas remote from the hemorrhages, denoting that the latter were not agonal but occurred before death. In the cortex, subcortical white substance, medulla oblongata and upper cervical cord they were absent.

The ganglion cells were universally changed aside from the phenomena of chromatolysis; the great majority, large and small, appeared pale, swollen, liquefied and frequently as mere shadows. Their nuclei were also pale, con-

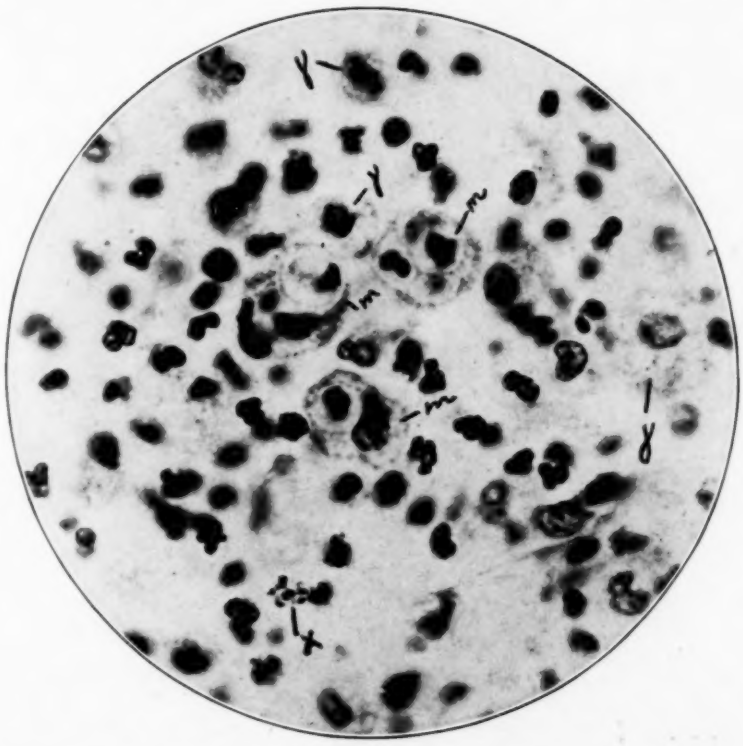

Fig. 4.—Optic thalamus. The infiltrating cells are numerous polymorphonuclear cells, macrophages (*m*) and gitter cells (γ); at *x* "minute bodies" are visible. Toluidin blue, × 800.

taining only a few granules of chromatin. In many instances they stained much better than the cytoplasm, and therefore appeared as numerous free round bodies, surrounded by pale remnants of broken up cytoplasm. Phenomena of satellitosis and neuronophagia, though present, were not conspicuous. It is noteworthy that the neuronophages were often polymorphonuclear leukocytes. Figure 6, for instance, shows markedly changed ganglion cells, each containing a vacuole with a polymorphonuclear cell within. The polymorphonuclear cells can be seen in the same picture covering the parenchyma, mixed with glia cells, while in figure 7, the field is entirely monopolized by glia cells. Like the vascular changes, those of the ganglion cells were especially in evidence

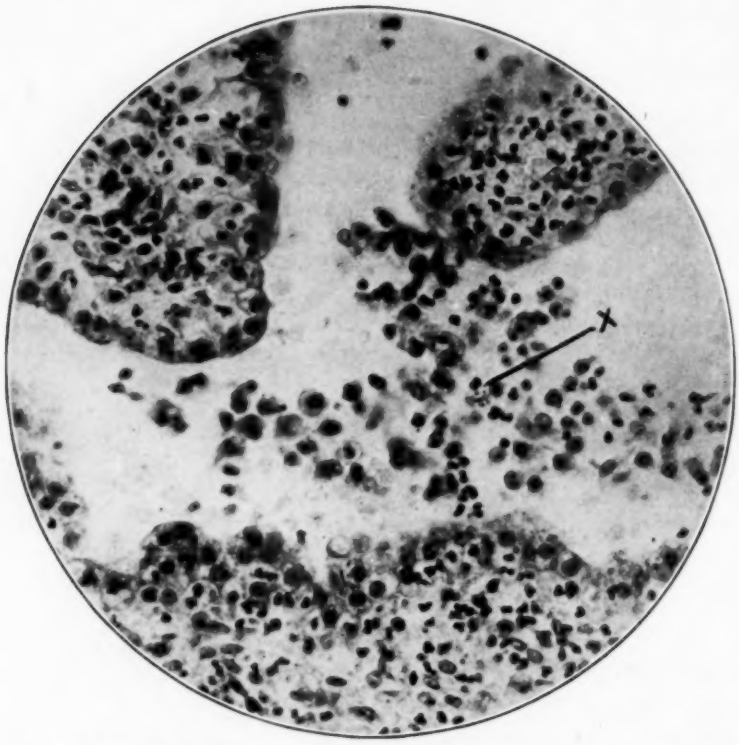

Fig. 5.—Choroid plexus. Polymorphonuclear and other cells described in the text are seen in the stroma as well as in the ventricles themselves. *x* indicates "minute bodies." Toluidin blue, × 300.

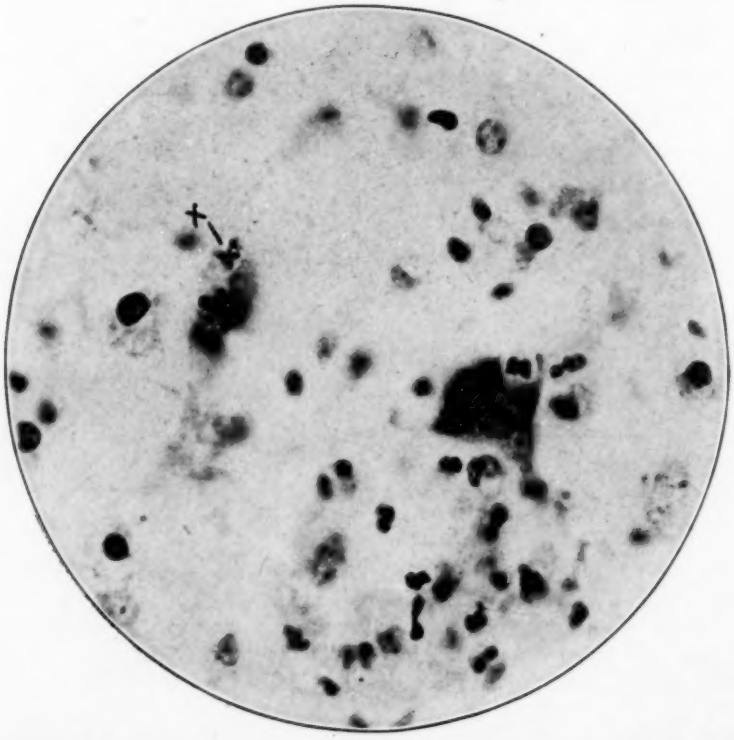

Fig. 6.—Optic thalamus. Two ganglion cells containing polymorphonuclear leukocytes within vacuoles; at *x* are "minute bodies." Toluidin blue, × 650.

in the pons and the basal ganglia, less so in the medulla and cervical cord. Here phenomena of central chromatolysis were the only ganglion cell changes.

Glia: In contrast to the ganglion cells, which exhibited advanced regressive changes, the glia showed universal progressive phenomena. In specimens stained with toluidin blue, Alzheimer-Mann stain and gold corrosive sublimate of Cajal, the glia cells appeared proliferated and hypertrophied (fig. 8). Especially was this in evidence in the marginal areas. The microglia cells, even with the toluidin blue staining method, appeared hypertrophied, their processes unusually well developed and visible; in short they showed as typical "Stäbchen-

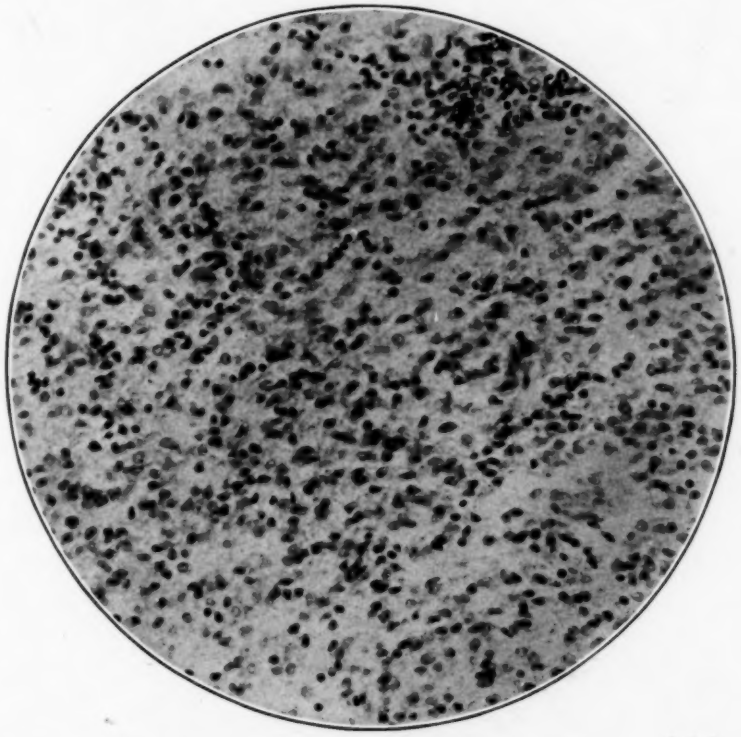

Fig. 7.—Optic thalamus. An area adjacent to the ependyma (*ep.*) is covered by a mass of glia cells; polymorphonuclear cells or minutes bodies are not present. Toluidin blue, × 230.

zellen" of Nissl. However, their number was not increased, nor did they show any signs of transformation into gitter cells, while in severely injured areas, such as those covered by hemorrhages, they were absent altogether. The glia nuclei or oligodendroglia, so numerous in the white substance, also appeared well preserved — their chromatin was abundant and, as a rule, a thin rim of cytoplasm could be discerned around the nucleus.

Meninges and Choroid Plexus: In some areas, such as around the occipital lobe and cerebellum, the meninges were moderately infiltrated; in other regions — frontoparietal (fig. 9) and temporal lobes — the infiltration was marked. The infiltrating cells were similar to those in the spaces of Virchow-Robin: poly-

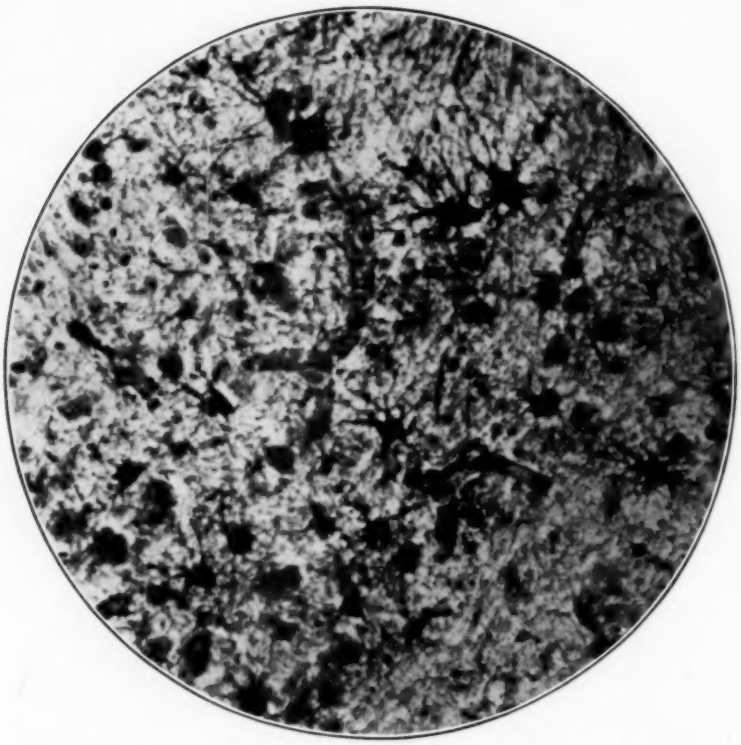

Fig. 8.—Pons. Cytoplasmic glia or macroglia. Cajal's gold corrosive sublimate stain, × 325.

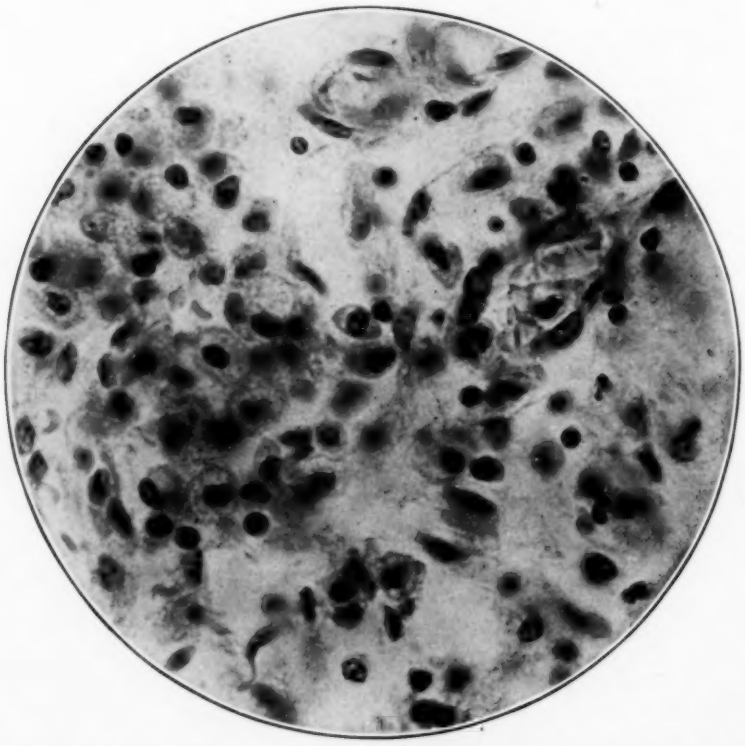

Fig. 9.—A high magnification of the meningeal infiltration cells. Polymorphonuclear leukocytes are few; instead there are numerous gitter cells and other formations described in the text. Toluidin blue, × 560.

morphonuclear leukocytes, lymphocytes, fibroblasts, occasional plasma and gitter cells and, in addition, large macrophages harboring blood pigment, broken up lymphocytes and other enclosures. Some of the infiltrating cells, especially the gitter cells and macrophages, are shown in figure 9 (occipital lobe). In some areas — motor, for instance — the infiltrating cells were dense, filling up the subarachnoid space (fig. 10) and following the intracerebral pial prolongations, but not invading the parenchyma. The choroid plexus was also greatly changed. The stroma (fig. 5) was covered by a mass of hematogenous elements, of the same type as found in the perivascular spaces and in the meninges

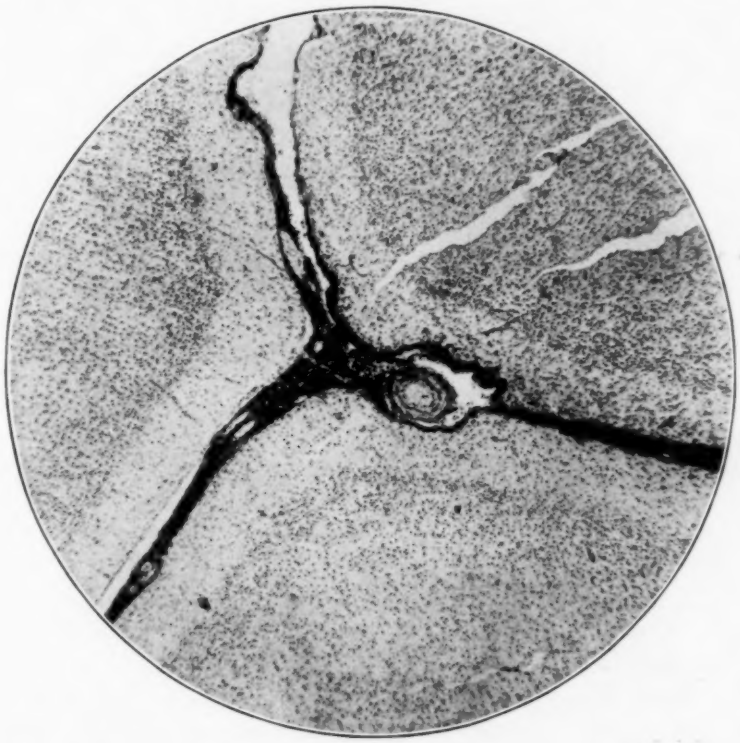

Fig. 10.—Parietal cortex. Meningitis, with an artery slightly infiltrated. The types of infiltrating cells are pictured in figure 9. Toluidin blue, × 42.

(lymphocytes, polymorphonuclear leukocytes, polyblasts, large plasma cells and numerous fibroblasts). Some cells, such as polymorphonuclear leukocytes and lymphocytes, were outside the plexus, within the ventricular cavity (fig. 5) almost adjacent to the tuft cells. The latter were for the most part granular and opaque and were often of huge size; the nuclei were pyknotic; the cell bodies were vacuolated and harbored, in some instances, enclosures, such as fragments of the hematogenous cells mentioned above. Occasionally the tuft cells appeared replaced by large round vacuolated bodies (fig. 11) containing enclosures, veritable macrophages. However, many tuft cells were practically normal. The ependymal cells were granular, large and often proliferated.

SUMMARY AND COMMENT

The changes outlined may be classified as belonging to an acute meningo-encephalitis. The unusual features were the extreme youth of the patient and the type of the inflammatory elements. Kemkes and Saenger [1] analyzed seventy-two cases of encephalitis in children. Of these, twelve cases were of the epidemic type; seventeen of questionable etiology; ten were postinfectious, and thirty-two of unknown etiology.

Fig. 11.—Choroid plexus. Macrophages with enclosures crowding and almost obliterating the tuft cells. Toluidin blue, × 560.

Twenty of the seventy-two cases occurred during the first year of life, and thirteen in the second year; hence encephalitis is a common disease in early childhood. The youngest patient, according to Hofstadt,[2] was aged 11 weeks; recovery followed an illness of four-

1. Kemkes, B., and Saenger, S.: Ueber Encephalitis des Kindes, Monatschr. f. Kinderh. **32**:334 (June) 1926.

2. Hofstadt, F.: Zur Kenntniss der Encephalitis epidemica im Kindesalter, Ztschr. f. Kinderh. **29**:191, 1921.

teen days' duration. Hoobler's [3] patient was still younger, 3 weeks of age, and also recovered, while Bassoe and I [4] reported a case of clinical and pathologic epidemic encephalitis in an infant, aged 4 weeks.

The case under discussion thus concerns the youngest patient in whom a meningo-encephalitis has been studied pathologically. Unfortunately, from the clinical information, it is not possible to determine the exact age at which the disease set in, but this certainly was younger than 15 days—the age at which the child died. Such an assumption is justified by the pathologic observations. Of these, the presence of the polymorphonuclear elements was especially striking. In the pons and optic thalamus they were predominant; in other regions they were less so. According to Häuptli,[5] three days after the onset of poliomyelitis, the infiltrating cells are almost all of polymorphonuclear type; in epidemic encephalitis, they predominate during the first eight or nine days. Levaditi, Harvier and Nicolau [6] remark that when the infection is severe the infiltrating cells are exclusively polymorphonuclear leukocytes; mononuclear infiltration, in contrast, corresponds in man to the chronic phase of the disease (epidemic encephalitis). From the foregoing, one may conclude that this case deals with an acute inflammatory condition of the brain of several day's duration and that the disease probably set in when the child was 1 week old.

The nature of the encephalitis in this case, as pointed out elsewhere,[7] is exceedingly difficult to determine from the type of the reactive inflammatory phenomena; that is to say, it is hard to tell whether one is dealing in any given case with an encephalitis, a cerebral form of poliomyelitis or a similar condition. Unfortunately, in this case only a small portion of the spinal cord could be studied. Here the ganglion cell and other changes were much milder than in the brain stem, a fact that would speak against poliomyelitis. Absence of a similar infection in the other twin, the localization of most striking changes in the midbrain and basal ganglia, the comparative mildness of ganglion cell destruction in the spinal cord, the regressiveness of the changes cephalad and caudad to the midbrain would suggest epidemic encephalitis. However, cases

3. Hoobler, B. R.: Encephalitis in the New-Born, Arch. Pediat. **38**:461 (July) 1921.

4. Bassoe, P., and Hassin, G. B.: A Contribution to the Histopathology of Epidemic ("Lethargic") Encephalitis, Arch. Neurol. & Psychiat. **2**:24 (July) 1919.

5. Häuptli, O.: Zur Histopathologie der Poliomyelitis acuta und der Encephalitis epidemica, Deutsche Ztschr. f. Nervenh. **71**:1, 1921.

6. Levaditi, G.; Harvier, P., and Nicolau: Étude expérimentale de l'encéphalite dite lethargique, Ann. de l'Inst. Pasteur **36**:63, 1922.

7. Hassin, G. B.: A Note on the Comparative Histopathology of Acute Anterior Poliomyelitis and Epidemic Encephalitis, Arch. Neurol. & Psychiat. **11**:28 (Jan.) 1924.

of acute epidemic encephalitis in man, with prevalence of polymorphonuclear leukocytes in the parenchyma, perivascular spaces and within the ganglion cells, are exceedingly rare. In my pathologic studies of numerous cases of this disease, I have never encountered such a condition. Neither did I find in the literature anything resembling it, except possibly the case of DaFano and cases of epidemic encephalitis produced experimentally in rabbits, monkeys and guinea-pigs.

DaFano's [8] patient was a man, aged 43, who "suddenly complained of headache and became unconscious." Death occurred thirty-six hours after the onset. The necropsy revealed hemorrhages in the subcortical white matter, perivascular and parenchymal infiltrations with "polymorphocytes," gitter cells and lymphocytes and the presence of so-called minute bodies. The infiltrations were marked in the upper portion of the medulla oblongata, pons, midbrain, optic thalamus and cortex, particularly in the frontal and parietal ascending convolutions. In the spinal cord (cervical region) only a slight small-cell infiltration could be detected. There also was an "active penetration of the polymorphocytes into the cytoplasm of the nerve cells." [9] As much as these observations resembled mine, those found in the experimental form of epidemic encephalitis were almost identical. Thus Levaditi and Harvier [10] describe the changes as follows: the perivascular infiltrating cells (p. 963) are both polymorphonuclear and mononuclear; in the meninges and the perivascular spaces they may be exclusively mononuclear, but the "encephalitis itself is engendered by the polymorphonuclears," which is not observed in man; neuronophagia is rare (in rabbits); the spinal cord is not affected (in guinea-pigs), though the changes in monkeys, rabbits and guinea-pigs are practically alike. The experimental type of epidemic encephalitis is much richer in pathologic conditions than the human type, which is probably the result of the infection being more severe and acute, the duration of the disease shorter, and of other factors. Aside from the frequent occurrence of polymorphonuclear leukocytes in experimental encephalitis, hemorrhages are also common, but they are exceptionally rare in the human form. The prevalence or the mere presence of hemorrhages may suggest a type of acute encephalitis described as hemorrhagic. I am not in a position to discuss this form,

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8. DaFano, C.: The Histology of the Central Nervous System in an Acute Case of Encephalitis Presumably Epidemic, J. Path. & Bacteriol. **27**:11, 1924.

9. Since this paper was written, Dr. Johan Eckel's contribution, "Encephalitis Acutissima," has appeared in *Jabrb. f. Psychiat. u. Neurol.*, October, 1926. His patient was a man, aged 50, who died two hours after the onset of the symptoms. Histologic observations revealed marked leukocytic infiltrations of the perivascular spaces especially in the pons, brain stem and less so in the cortex.

10. Levaditi, G., and Harvier, P.: Étude expérimentale de l'encéphalite dite lethargique, Ann. de l'Inst. Pasteur **34**:9 (Dec.) 1920.

as I do not know whether such a pathologic entity exists and as I have not had any personal experience with it. Neither am I in a position to speak of so-called influenzal encephalitis in which hemorrhages are said to be frequent and abundant. In the case reported here the hemorrhages were strictly focal, occurring only in the pons and the right lenticular nucleus. They may indicate not so much a specific form of encephalitis as the gravity of the infection that caused it. However, they were not accidental or agonal, as the phenomena of the reactive glia show that they occurred before death.

I should like to emphasize one more point—the presence of the granules, pictured in figures 4, 5 and 6. They were described by DaFano [8] in his case as "very small structures, or rather small granules, of somewhat variable size and shape. In most instances they occurred singly, but now and then also in pairs and surrounded by a clear halo. They were confined to the cytoplasm of the polymorphocytes or adherent to the edges of the cells and were never observed with certainty outside the cells in the nerve substance. In a few instances almost identical granules were noticed within lymphocytes, but they were not taken into account, being insufficiently defined, particularly when seen under very high magnification." In previous communications DaFano described "minute bodies" within the ganglion cells in epidemic encephalitis and suggested that they might be the result of the presence in the tissues of a living agent—the cause of the disease.

On the basis of the pathologic studies of this case, of that of DaFano and the facts furnished by experimental research, one may conclude that the case here recorded was most likely one of epidemic encephalitis, that is to say, an exogenous infectious disease. The condition affected a new-born child, one of twins, and was not inborn. Had the child recovered, with the disease unrecognized or not even suspected, some sequelae such as epilepsy, mental deficiency, idiocy or behavior disorder would be expected. In other words, any of these or similar disorders observed in grown children may be caused by inflammatory brain lesions occurring in early infancy.

A COMPARISON OF THE VERNES AND WASSERMANN TESTS AS APPLIED TO THE SPINAL FLUID*

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The nonspecificity of the Bordet-Wassermann test has stood as a challenge to workers in the field of immunology since its introduction. The desire to substitute for it a direct method that would dispense with the hemolytic system has furnished the impetus for much work by immunologists, but complete success has not crowned these efforts; the substitute methods have not proved to have more practical value than the method they have sought to supersede.

Shortly after the introduction of the Wassermann test, Michaelis [1] described a precipitation test for syphilis in which he employed an aqueous extract of syphilitic liver. When this was mixed with the blood serum of a syphilitic person, a visible precipitate resulted. Numerous investigators [2] followed Michaelis with proposals for diagnostic tests based on the formation of visible precipitates in mixtures of syphilitic serum and tissue extracts. Jacobstahl [3] was the first to publish a method that utilized the ultramicroscope for the recognition of the precipitate. All of these methods are based on the fact that mixtures of syphilitic

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* This work was made possible through a grant from the Commonwealth Fund.

1. Michaelis, L.: Precipitinreaktion bei Syphilis, Berl. klin. Wchnschr. **44**:1477, 1907.

2. Klausner, E.: Vorlaufige Mitteilung über eine Methode der Serum-diagnostic bei Lues, Wien. klin. Wchnschr. **21**:214, 1908. Porges, O., and Meier, G.: Ueber die Rolle der Lipoide bei der Wassermannschen Syphilis-Reaktion, Berl. klin. Wchnschr. **45**:731, 1908. Bruck, C., and Hidaka, S.: Ueber Fallungserscheinungen beim Vermischen von Syphilisseren mit alcoholischen Lueslеberextrakten, Z. Immunitätsforsch., Orig. **8**:476, 1911. Herman, O., and Perutz, A.: Med. Klin. **7**:60, 1911. Hecht, H.: Wassermannsche Reaktion und Präzipitation, Z. Immunitätsforsch., Orig. **24**:258, 1915-1916. Meinicke, E.: Ueber eine neue Methode der serologischen Luesdiagnose, Berl. klin. Wchnschr. **54**:613, 1917. Sachs, H., and Georgi, W.: Zur Serodiagnostik der Syphilis mittels Ausflockung durch cholesterinierte Extrakte, Med. Klin. **14**:805, 1918. Dreyer, G., and Ward, H. K.: A Simple Quantitative Serum Reaction for the Diagnosis of Syphilis, Lancet **1**:956, 1921. Dold, H.: Eine vereinfachte, frühzeitig makroskopische ablesbare Luesflockungsreaktion, Med. Klin. **17**:940, 1921. Bruck, C.: Beitrag zur Serodiagnose der Syphilis durch eine neue Methode, München. med. Wchnschr. **69**:569, 1922.

3. Jacobstahl, E.: Versuche zu einer optischen Serodiagnose der Syphilis, Z. Immunitätsforsch., Orig. **8**:107, 1910-1911.

serum and alcoholic extracts of animal organs will produce visible precipitates when the relative proportions of the two ingredients are proper and sufficient incubation is used.

Work along this line has accomplished enough during the last few years to justify the belief that eventually some direct method of flocculation or precipitation will replace the present round-about procedure of arriving at the diagnosis of syphilis by complement fixation. The published reports indicate that the direct flocculation method of Kahn [4] closely parallels the complement fixation test of Bordet-Wassermann in active untreated syphilis but divergences occur when the tests are compared in treated syphilis.

Most of the conclusions that have been drawn from comparative studies appear to be based on the assumption that the Bordet-Wassermann result should be the index of accuracy, therefore variations from it are interpreted as indicating a diminished specificity or sensitivity of the test that is used for comparison. That this is a false standard is obvious. The best standard is examination of the microscopic changes, and, in the absence of that, clinical observation.

In a series of consecutive studies from 1909 to 1922, Vernes,[5] step by step, devised a flocculation test for syphilis that is susceptible of rigid standardization. In 1917, he first reported his observations concerning the precipitating effect of blood serum on a mineral colloid (ferric hydrate) and demonstrated that normal blood serum and syphilitic blood serum differ in respect to the periodicity of the flocculation phenomenon. The difference could be detected when mineral substances other than ferric hydrate were used. After testing numerous organic and inorganic substances, he concluded that mineral substances, when converted into the colloidal state, were too variable in their physical properties to fulfil the requirements of a diagnostic test for syphilis. He therefore gave

4. Kahn, R. L.: A Simple Quantitative Precipitation Reaction for Syphilis, Arch. Dermat. & Syph. **5**:570 and 734 (May) 1922; **6**:332 (Sept.) 1922; Serum Diagnosis of Syphilis by Precipitation, Baltimore, Williams and Wilkins Co., 1925.

5. Vernes, A.: Les signes humoraux de la syphilis. Introduction à l'étude des conditions expérimentales du traitment de la syphilis, Thèse de la doctorat, Paris, 1913; Qu'est la séro-réaction de la syphilis? Presse méd. **25**:704, 1917; Sur la précipitation de l'hydrate de fer colloidal par le sérum humain normal ou syphilitique, Compt. rend. Acad. d. sc. **165**:769, 1917; Sur la précipitation d'un colloïde organique par le sérum humain ou syphilitique, ibid. **166**:575, 1918; De la mésure colorimétrique de l'infection syphilitique, ibid. **167**:383, 1918; Indice syphilimétrique, détermination colorimétrique des ecartes de stabilité, ibid. **167**:500, 1918; Le graphique du syphilitique, ibid. **168**:247, 1919; Qu'est la séro-reaction de la syphilis?, Presse méd. **27**:333, 1919. Cornwall, L. H., and Aronson, L. S.: Phenomenon of Vernes as Applied to the Serodiagnosis of Syphilis, J. A. M. A. **75**:1697 (Dec. 18) 1920. Cornwall, L. H.: Further Experiences with the Vernes Test for the Diagnosis of Syphilis, J. Lab. & Clin. Med. **10**:831, 1925.

his attention to organic extracts and finally chose horse heart as the most suitable tissue. The extract that is used for the flocculable colloidal suspension consists of the alcohol soluble residue from horse heart after removal of all lipoidal substances that are soluble in ethylene perchloride. The muscular portion is washed with 96 per cent alcohol, dried and pulverized. This is then extracted in a Soxhlet apparatus with ethylene perchloride ($C_2H_4Cl_2$). The residue is allowed to dry and is then extracted with 96 per cent alcohol. This is quantitatively standardized so as to contain 15 Gm. of dried extract per liter of alcohol.

A colloidal suspension of the alcohol soluble derivatives from horse heart is prepared by mixing the extract with distilled water. This mixture is always prepared at the same temperature (25 C.). The addition of distilled water to the extract or vice versa is always made at a constant speed by means of a pipet devised to deliver its contents at a given rate per minute. The ingredients are mixed at a given speed by means of a glass propeller operated by a motor, regulated by a rheostat and equipped with a gag showing the number of rotations per minute.

By these methods of precision colloidal suspensions are obtained that have similar physical states. The constancy of the physical character of colloidal suspensions so prepared may be confirmed by measuring the diffusion with the photometer of Vernes, Bricq and Yvon. The rest of the technic consists of mixing the suspension with blood serum or spinal fluid and incubating this at 25 C. for four hours.

The result, in the case of syphilitic serum or fluids, is a flocculation of the colloidal particles out of suspension which is accompanied by an alteration in the turbidity. This alteration is too delicate for macroscopic detection and is therefore read by means of a photometer. The readings are made directly from an illuminated scale in the instrument and are in terms of optic density.[6] The optic density of water being 0,

6. Density was chosen as the optic characteristic for the measure of flocculation because of its convenience. It is measured by means of a photometric wedge of smoked glass on which there is arranged a graduated scale. The light enters the apparatus in parallel rays. This is accomplished by means of an ordinary condenser. After entering the apparatus, the light is separated into two bands, one of which passes through the fluid to be examined and the other through a wedge of smoked glass. The purpose of the latter is to equalize the two bands by absorbing the same amount of light as the fluid under examination.

These two bands are later brought together so as to form a circular field with a central band. The central band represents the light that has passed through the photometric wedge, and the rest of the circle, consisting of two semicircular fields, represents the light that has passed through the liquid under examination. The absorption of light by the wedge is accomplished by increasing the thickness of the interposed portion. The wedge is manipulated so that

any increase is expressed numerically in hundreths from 0 to 1.50 or more. Optic density is the measure of light absorption or diffusion and is arrived at by determination of the logarithm of the relation of the incident light ray and the transmitted light ray. The lowest readings are 0 or less, and the highest readings may exceed 1.50. The numerical readings of optic density cannot be translated into the present serologic terminology, and an attempt should not be made to do this. The readings of the photometric scale may, however, be converted into volumetric terms and expressed in milligrams of precipitate per cubic centimeter.

According to the accumulated experience to date, readings greater than 0.27 always mean syphilis. In the personal experience of Vernes,[7] a reading above 0.27 has never been obtained with a nonsyphilitic serum or spinal fluid. One cannot say that readings lower than 0.27 always mean the absence of syphilis, because all blood serums and spinal fluids may produce flocculation under certain conditions and the flocculation of the blood serums and spinal fluids of normal individuals is subject to variation from time to time. The upper limit of normal flocculation having been established as 0.27, all readings above this point indicate hyperflocculence due to syphilis. The technic has been adjusted so that the flocculence given by normal or nonsyphilitic serum is confined to the lower degrees of the scale.[8]

The most recent conclusions of Vernes [9] and his co-workers concerning the interpretation may be summarized as follows: 0 or less indicates

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the intensity of the band of light traversing it is equal to the intensity of the other band which has traversed the tube containing the blood serum, spinal fluid or other material.

The scale on the photometric wedge is illuminated by a small electric bulb, and the reading is made with a magnifying lens that is inserted through an aperture in the apparatus (figs. 1 and 2).

The scale is verified each time the photometer is used by checking it with a light filter, the absorptive index of which is known. If necessary it is then adjusted so that its reading corresponds to the known absorptive index of the interposed filter. For a full description of this apparatus, the reader is referred to the papers dealing more directly with technic given in footnotes 5, 7 and 9, and in Baylis, A. B., Sheplar, A. E., and MacNeal, W. J.: Simultaneous Vernes and Wassermann Tests for Syphilis, Arch. Dermat. & Syph. **10**:306 (Sept.) 1924; The Vernes Flocculation Test for Syphilis, ibid. **12**:242 (Aug.) 1925; The Vernes Flocculation Test as an Accessory Serologic Guide in the Combat Against Syphilis, Am. J. Syph. **10**:298, 1926.

7. Vernes, A.: Travaux et publications de l'institute prophylactique, Fascicule 2, Paris, A. Maloine et Fils, 1923.

8. Considerable misunderstanding of results and misinterpretation of their significance will arise if attempts are made to draw analogies between degrees of optic density and the plus signs that are employed as expressions of complement fixation. Such efforts will not serve a useful purpose in an understanding of this reaction.

9. Vernes, A.: Travaux et publications de l'institute prophylactique, Fascicule 3, Paris, A. Maloine et Fils, 1924.

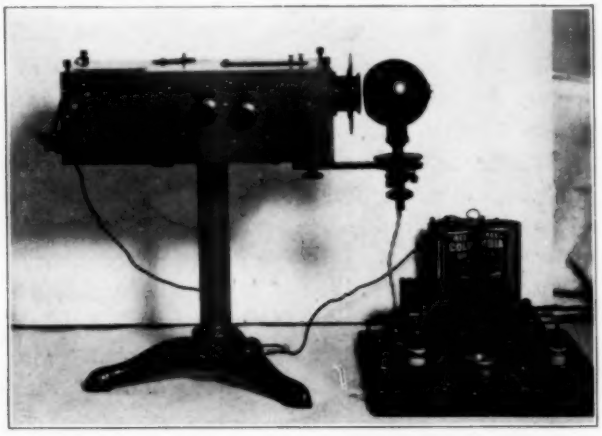

Fig. 1.—Photometer of Vernes, Bricq and Yvon.

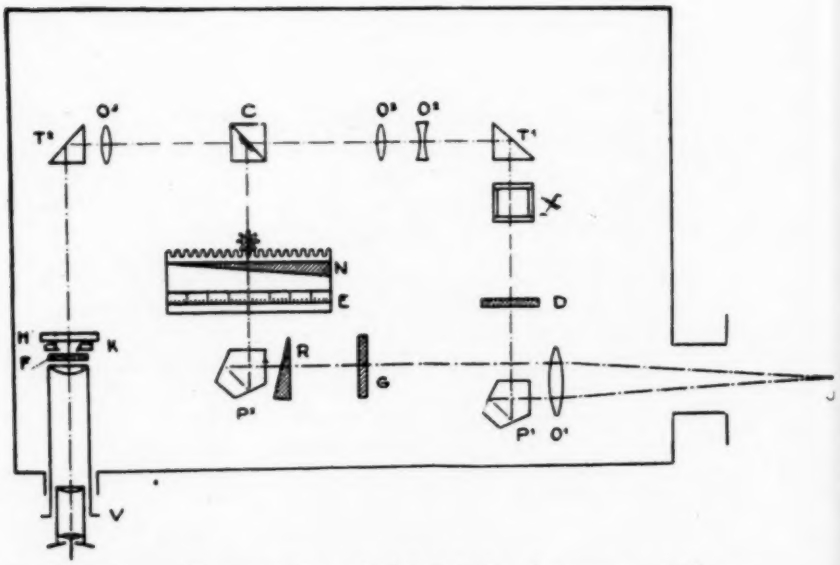

Fig. 2.—Diagram to illustrate the principle of the photometer of Vernes, Bricq and Yvon. One band of light passes through cube *X* containing the fluid to be tested; the other passes through the movable wedge, *N*. *E* is the scale from which the results are read.

a completely normal serum; 0.01 or 0.02 suggests that the serum is normal but is regarded with some suspicion; 0.03 or 0.04 occurs in about 2 per cent of normal serums, and among 100 serums, selected at random, that give this density approximately 25 per cent will be syphilitic and 75 per cent nonsyphilitic; 0.05 or 0.06 may occur in 0.2 per cent of normal serums (1 in 500) and among 100 serums, selected at random,

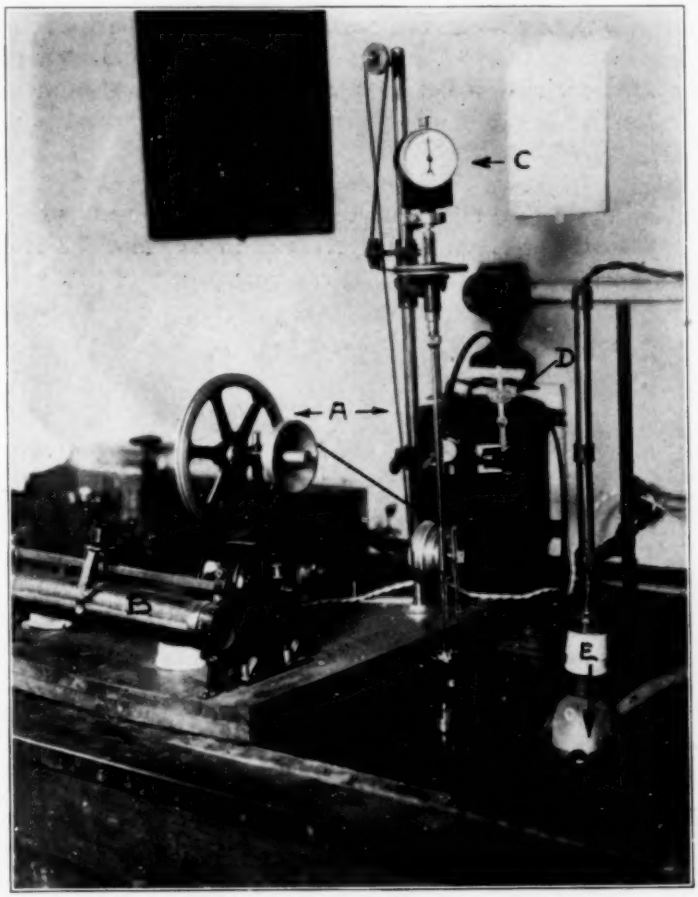

Fig. 3.—Mixing apparatus (*A*) with rheostat (*B*), revolution counter (*C*), special pipet (*D*) and water bath (*E*).

that give this density 50 per cent will be syphilitic and 50 per cent nonsyphilitic; among 2,000 serums giving the density of from 0.07 to 0.11, only one will be nonsyphilitic; among 10,000 serums giving the density of from 0.12 to 0.18, only one will be nonsyphilitic; among 650,000 serums giving the density of 0.27, only one will be nonsyphilitic.

It is obvious from the foregoing that more precise standards should be established for each point of the scale from 0 to 0.27. For instance,

if the chance of 0.06 being syphilitic or nonsyphilitic is equal, then the density 0.07 could scarcely represent the syphilitic probability of only 1 in 2,000. When the test gains a wider range of application further modifications will undoubtedly result.

In nonsyphilitic cases the flocculence of the blood serum at successive examinations varies only slightly and the graph displays a horizontal line or one with slight oscillations. In frank and unquestionable syphilis the graph illustrating successive examinations displays the effectiveness of the treatment on the surflocculence of the blood serum. Progression, stability and regression of the infection are reflected in the rise, stationary character or fall of the photometric readings.

MATERIAL

This report is concerned with a comparison of the results of Vernes and Wassermann tests on 490 specimens of spinal fluid from 200 patients selected from the Neurological Department of the Vanderbilt Clinic. In sixty-two cases repeated examinations of the spinal fluids were made, the largest number being twenty. In the remaining 138 cases, only one fluid examination was made.

TABLE 1.—*Neurologic Status of Cases*

	Cases	Fluids
1. Mesodermal (meningovascular) neurosyphilis	72	186
2. Ectodermal (parenchymatous) neurosyphilis	65	224
3. Mixed neurosyphilis	16	32
4. Non-neurologic syphilis	9	9
5. Nonsyphilitic	38	39
	200	490

On the basis of the neurologic status the cases were separated into the groups given in table 1.

The diagnosis of neurosyphilis was based on a history of syphilitic infection or positive serologic observations associated with positive evidence of organic disease of the nervous system that could not be satisfactorily explained otherwise. The non-neurologic syphilitic group included cases that gave a history of syphilitic infection but in which organic neurologic disease was not present. The nonsyphilitic group consisted of cases in which a history of syphilis could not be obtained and in which a clinical basis for that diagnosis from the neurologic observations did not appear.

An endeavor has been made to correlate these laboratory observations with the clinical aspects of the cases in the hope that the comparative significance of the two tests may be evaluated. The subdivision of neurosyphilis into mesodermal, ectodermal and mixed types was determined by correlation of the clinical features and laboratory results. The latter included a record of color, pressure, cells, globulin, Wassermann, Vernes and colloidal gold tests.

MESODERMAL (MENINGOVASCULAR) NEUROSYPHILIS

Mesodermal neurosyphilis is regarded as that form in which the clinical features indicate that the predominant pathologic change is in or immediately adjacent to the structures of mesodermal origin, namely, the meninges and the blood vessels. Pathologically, it is manifested by exudative or productive lesions. The spinal fluid observations are subject to certain variations depending on the acuity or chronicity of the process. The cell count is the factor of chief importance in determining the acuity of the process. The globulin is always increased unless the disease is confined exclusively to the vascular system or the process is completely arrested. The Wassermann reaction is positive in the spinal fluid in nearly all cases unless the process is arrested or limited to the vascular system. The colloidal gold curve usually shows maximum precipitation in the third to the fifth tubes, the change being represented by the numbers 3 or 4, e. g., 1123321100.

There were seventy-two cases of mesodermal neurosyphilis. The total number of fluid examinations was 186. Thirty patients had more than one fluid examination, and there was agreement between the two tests in twenty-nine (96.6 per cent).

The case in which the two tests were in disagreement was one of long standing infection in a man, aged 53. The primary infection had occurred thirty years previously. Treatment was pursued for four years following the initial lesion and consisted of medication by mouth. In 1916, right external ophthalmoplegia with ptosis, external strabismus and pupillary dilatation developed. Treatment, consisting of arsphenamine, mercury and intraspinal injections, was instituted at that time. The blood Wassermann reaction always remained positive. A Vernes test on the blood was made twice and was negative on each occasion. Both tests were performed on the spinal fluid three times. On each occasion the Vernes test was negative and the Wassermann test was four plus in 2 cc. The cells varied from 1 to 8, the globulin from negative to one plus, and the colloidal gold from 4422332100 to 1111000000. The treatment since 1918, when the patient first came under the care of the clinic, has consisted of thirty intravenous injections of arsphenamine, almost continuous mercurial administration, two intraspinal injections by the Swift-Ellis technic, and two spinal drainages. Clinically, this case has remained stationary, but the blood and spinal fluid Wassermann reactions are positive and the Vernes reactions negative. The results of the Vernes test conform to the clinical status of this case in every sense as satisfactorily as do the results of the Wassermann test.

In forty-two cases in which only a single fluid examination was made, there was agreement in thirty-four (80.9 per cent). In six, the Wassermann reactions were positive and the Vernes reactions negative. With the exception of one case with a count of thirty, the cells were less than ten and the globulin moderately increased. The fluid with thirty cells gave an elevated colloidal gold curve (4455555554). In the other cases the colloidal gold curve was normal or low. Five of these cases were con-

sidered to represent generalized vascular syphilis of which the neurologic symptoms were part. The fifth case, the one showing thirty cells, represented one of generalized dissemination of the spirochetes early in the disease, and in the light of other experiences it is the belief that the negative result indicated a low virulence of the infecting organism and bespoke a good prognosis.

Two other cases in which only one fluid examination was made resulted in negative Wassermann and positive Vernes tests. Both patients were in the fifth decade of life. One gave a history of syphilitic infection twenty-four years previously, with intermittent treatment since that time. Subjective symptoms were not present, and the objective symptoms consisted of some reflex inequality and a unilateral Argyll Robertson pupil. The other patient did not give any history of syphilitic infection and presented a typical parkinsonian syndrome. The blood was positive with both tests in the former, but in the latter the blood Wassermann reaction was positive and the Vernes negative (0.0). In both cases the fluid Wassermann reaction was negative and the Vernes reaction positive, 0.43 and 0.72, respectively.

The comparative results of these single examinations indicate the limitation of the Vernes method as a diagnostic test. When a quick decision is demanded, and this must be made on a single examination, my experience persuades me that a single positive result has definite value, but a single negative result is of slight significance. In a case of known syphilis, however, a negative Vernes reaction speaks in favor of a low grade of virulence and therefore of a hopeful prognosis for arresting the infection.

ECTODERMAL (PARENCHYMATOUS) NEUROSYPHILIS

Ectodermal neurosyphilis is considered to be that form in which the clinical symptoms reflect lesions in the neural components of ectodermal derivation: the neurons and the neuroglia. Pathologically, these lesions are primarily degenerative in type, in contrast with the exudative or productive features that characterize involvement of the mesoderm. The most typical examples of this form of the disease are to be found in the syndrome of general paralysis, tabes dorsalis and taboparesis.

The ectodermal group was represented by sixty-five cases with 224 examinations of the fluid. In twenty-seven cases more than one fluid examination was made and there was disagreement in two of these (7.4 per cent).

Both of these patients had tabes dorsalis. One had been treated steadily for two years with arsphenamine, mercury and intraspinal injections by the Swift-Ellis technic. The blood was negative to both tests. The spinal fluid cells, globulin and gold curve were normal. The Wassermann reaction was four plus in 2 cc. and two plus in 1 cc. on two occasions; the Vernes reactions were negative each time. Considering the stationary character of the disease in this case, results of the Vernes tests conform to the clinical status just as adequately, if not more so, than the Wassermann. The second case presented symptoms of involvement of the dorsal root characterized by lightning pains in

the extremities together with ataxia and Argyll Robertson pupils. On four occasions the fluid Wassermann reaction was negative in 2 cc. and the Vernes positive (1.24, 0.57, 0.53 and 0.38). On one occasion they were both negative and on another the Wassermann reaction was four plus in 2 cc. and the Vernes negative (fig. 7).

The results in these two cases strengthen one's regard for this new test. In the quiescent case it was negative when the Wassermann reaction was positive. In the active case it was positive when the Wassermann reaction was negative.

In thirty-eight cases of ectodermal syphilis in which only one examination of the fluid was made, there was divergence in six cases (15.8 per cent).

In two quiescent cases of tabes with negative blood reactions and normal cell counts and globulin and colloidal gold tests, the fluid Wassermann reaction was four plus in 2 cc. and the Vernes negative. In another quiescent case of tabes with a doubtful blood Wassermann reaction, 81 cells, two plus globulin, and an elevated gold curve (2223331100) in the spinal fluid, the Wassermann reaction was four plus with 0.2 cc. and the Vernes negative. A fourth case of tabes with both reactions positive in the blood showed a negative Wassermann and positive Vernes reaction (0.47) in the spinal fluid, 3 cells, normal globulin, and an elevated colloidal gold curve (5543211000). In this case the clinical evidence indicated a progressive pathologic change with gradual increase in the ataxia. The fifth case was one of encephalic syphilis with a paranoid trend, in which the cells, globulin, colloidal gold and Wassermann tests were negative but the Vernes test was positive (0.35). The sixth case was one of quiescent tabes with a negative blood Wassermann reaction, normal cells and globulin, and a negative colloidal gold and fluid Wassermann test, but with a positive Vernes reaction in the spinal fluid (0.21).

In these six cases, five of which were of tabes, the Vernes results were regarded as more in keeping with the clinical aspects in three, being negative when the Wassermann reaction was positive in two quiescent cases and positive when the Wassermann reaction was negative in a progressive case. In the other three cases the Wassermann reaction was regarded as more consonant with the clinical aspects.

MIXED NEUROSYPHILIS

The mixed group comprised those cases in which it was concluded from the clinical features and laboratory results that the pathologic change involved to an equal degree the meninges, blood vessels and parenchymatous structures. There were sixteen cases of this form of neurosyphilis with thirty-two examinations of the fluid. In five cases two or more examinations of the fluid were made and in eleven cases a single examination only. There were not any disagreements between the two reactions in this group of cases.

The comparative results of these two reactions in 153 cases of neurosyphilis are summarized briefly in table 2.

NONSYPHILITIC GROUP

Thirty-eight cases were considered clinically as nonsyphilitic on the basis of the history and physical observations. There was agreement in thirty (79 per cent) and disagreement in eight (21 per cent). A brief summary of the data concerning these cases follows:

CASE 1.—A man, aged 53, who did not give a history of syphilis and who had a negative blood Wassermann reaction, suddenly developed a right hemiparesis. The clinical diagnosis was cerebral thrombosis on an arteriosclerotic basis. The spinal fluid revealed normal cells and negative globulin and Wassermann tests. The colloidal gold curve was 5544321100. The Vernes reaction was positive (0.66).

CASE 2.—In a man, aged 48, with a spastic gait, increased deep reflexes, transient patellar clonus and permanent left ankle clonus, left Babinski sign, and diminution of touch and pain sensations in the left leg, the blood Wassermann and Vernes reactions were negative. The clinical diagnosis was disseminated lesions of the pyramidal system. The spinal fluid revealed normal cells, and the globulin, Wassermann and colloidal gold tests were negative. The Vernes test was positive (0.50).

TABLE 2.—*Comparative Results of Examinations of Neurosyphilitic Fluids*

	Fluids, Total Number	Cases		
		Total Number	Agreement	Disagreement
Two or more examinations..	351	62	59	3 cases 4.8%
Single examinations	91	91	77	14 cases 15.4%
	442	153	136	17 cases 12.5%

CASE 3.—A woman, aged 25, with normal organic symptoms, was diagnosed as "paranoid trend." The blood Wassermann reaction was negative but the Kahn test was positive. The spinal fluid revealed 5 cells; a positive globulin test; a negative Wassermann test, colloidal gold test, 1111000000, and Vernes test positive (0.36).

CASE 4.—A man, aged 38, denying knowledge of syphilitic infection, presented rapidly progressive bilateral optic atrophy of three months' duration. The only other positive organic symptoms were diminished patellar reflexes that could be elicited only on reenforcement. The spinal fluid did not reveal any cells, and the globulin, colloidal gold and Wassermann tests were negative. The Vernes test was positive (0.45).

CASE 5.—A woman, aged 35, presented a radiculitis of undetermined etiology. The blood Wassermann reaction was negative, and syphilis was denied. The spinal fluid revealed: 20 cells; one plus globulin; negative Wassermann test; colloidal gold test, 3332100000. The Vernes test was positive (0.27).

CASE 6.—A child, aged 4, presented symptoms of myelitis of undetermined etiology. The blood Wassermann reaction was negative. The spinal fluid was xanthochromic, with spontaneous coagulation; not many cells were present; globulin was four plus and the colloidal gold test, 0000000000; the Wassermann reaction was negative. The Vernes test was positive (0.54).

CASE 7.—A man, aged 42, denying syphilis, complained of persistent headache with a sensation of heaviness. There was some equilibratory ataxia and the patellar reflexes could be elicited only with reenforcement. The blood

Wassermann test was negative. The spinal fluid revealed 60 cells, and negative globulin, gold and Wassermann reactions. The Vernes test was positive (0.39). The clinical diagnoses were: diabetes, chronic catarrhal otitis media, anxiety psychoneurosis.

CASE 8.—A woman, aged 34, wife of a patient suffering from general paralysis, complained of occipital headaches and pains in the left shoulder and back. The pupils were miotic, unequal and sluggish in reaction to light. The right patellar reflex was absent and the left present only with reenforcement. Vibratory sensation was diminished in the hands and feet. The blood Wassermann and Vernes reactions were negative. The spinal fluid revealed 4 cells, 2 plus globulin, and negative gold and Wassermann reactions. The Vernes test was positive (0.39). The clinical diagnosis was incomplete tabetic syndrome.

These eight cases would appear to indicate that the Vernes results were in error. On the other hand, with the meager data obtainable and the lack of observation beyond the initial examination, final decision must be reserved. At least one of the patients (case 3) had another positive serologic symptom in the blood (positive Kahn reaction) and another (case 8) was the wife of a patient having general paralysis and presented clinically an incomplete tabetic syndrome.

RESULTS OF TESTS IN VARIOUS SYPHILITIC CONDITIONS

CASE 1 (fig. 4).—R., a man, aged 53, was referred to the Neurological Department of the Vanderbilt Clinic, May 26, 1919, with the diagnosis of chronic myocarditis and chronic aortitis. The patient denied knowledge of any syphilitic infection, but the blood Wassermann reaction was four plus. The subjective symptoms consisted of pain in the shoulders and over the precordial region. The neurologic examination did not disclose any organic lesions of the central nervous system. Examination of the spinal fluid revealed four cells; a slight globulin excess; a negative Wassermann reaction, and a colloidal gold curve, 0000000000. Treatment from May, 1919, to October, 1922, consisted of nineteen injections of neoarsphenamine (7.6 Gm.), six injections of silver arsphenamine (1.2 Gm.) and fifty-nine intramuscular injections of mercury (59 grains). At the end of this time, the Wassermann reaction in the blood was unchanged, and the Vernes photometric reading was 0.05. Between October, 1922, and December, 1923, the blood was tested on four occasions, the Wassermann result always being four plus and the Vernes photometric reading varying from 0.00 to 0.07. Clinical symptoms of active syphilitic infection were not present. The Vernes results indicated that the infection was arrested or cured. The Wassermann results were not different from those seen in active syphilis. Results of this kind illustrate the desirability of using methods other than the Wassermann and raise the old question whether a positive Wassermann serologic reaction alone is sufficient justification for the indefinite continuation of antisyphilitic therapy. The clinical status of this patient has not changed since 1919, a period of seven years, and evidence of active syphilitic infection is not present.

CASE 2 (fig. 5).—B., a man, aged 26, presented himself in June, 1924, with objective manifestations consisting of emotional instability showing impaired orientation for time, place and persons. There was marked impairment of memory with complete alteration in behavior from the previous condition. There was a history of syphilitic infection in 1916. The alteration in conduct began in

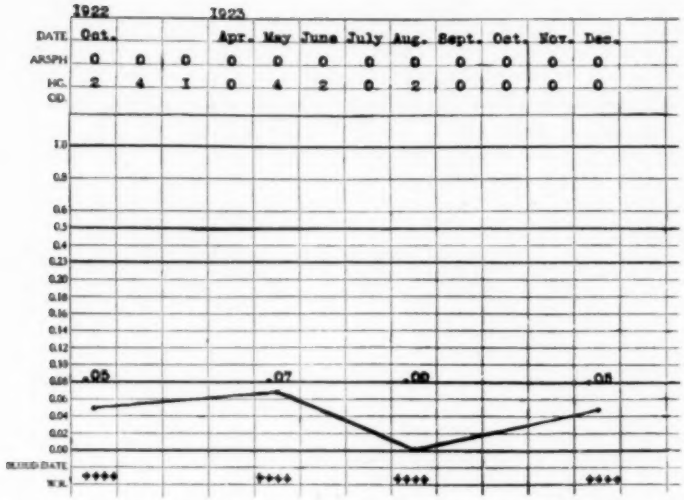

Fig. 4.—Comparison of Vernes and Wassermann reactions in patient R. The diagnoses were: etiologic — syphilis; anatomic pathologic — vascular; clinical — aortitis. In this and figures 5, 6 and 7, the date of the examinations and the amount of treatment is shown at the top of the charts. The spinal fluid observations are immediately below, the Vernes results in the spinal fluid being shown by dotted lines. The Vernes results in the blood serum are represented by a solid line. The Wassermann results in the blood serum are shown at the bottom of the charts.

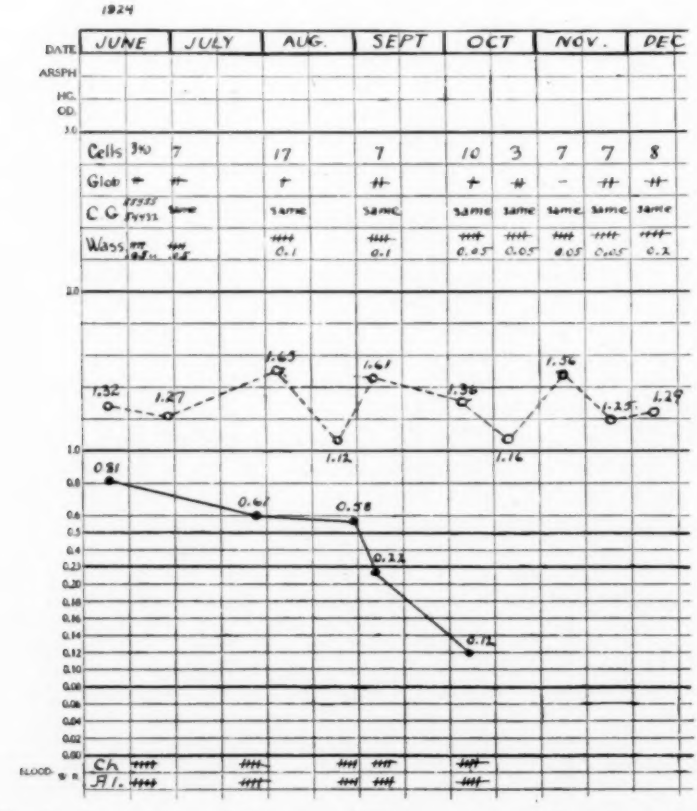

Fig. 5.—Comparison of Vernes and Wassermann reactions in patient B. The diagnoses were: etiologic — syphilis; anatomic pathologic — ectodermal; clinical — general paralysis.

February, 1923. The neurologic examination disclosed a positive Romberg sign, ataxia in the upper and lower extremities, present deep and superficial reflexes, Argyll Robertson pupils and a defect in speech of the general paralytic type. The clinical diagnosis was general paralysis. The Wassermann and Vernes reactions in both the blood and spinal fluid were positive. The treatment from June to December, 1924, consisted of twenty-three intravenous injections of silver-arsphenamine (5 Gm.), three of neoarsphenamine (1.8 Gm.), twenty-three intramuscular injections of mercury (23 grains) and thirteen intraspinal injections. There was little alteration in the serology of the blood or spinal

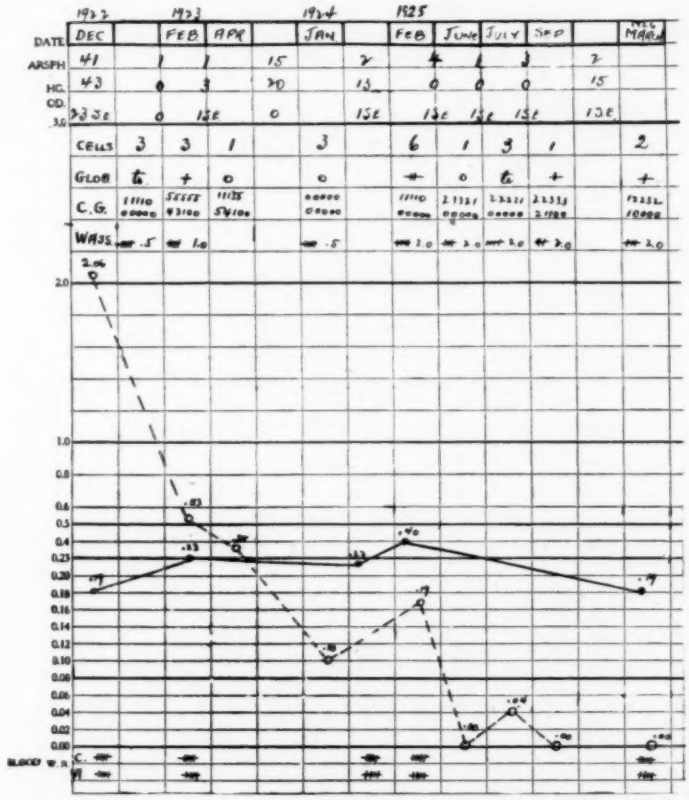

Fig. 6.—Comparison of Vernes and Wassermann reactions in patient B. The diagnoses were: etiologic — syphilis; anatomic pathologic — mixed; clinical — meningomyelitis.

fluid with the exception of a gradual drop in the Vernes readings in the blood. Improvement in the clinical symptoms did not take place during the same period. Here an unequivocal active and progressive syphilitic infection was being dealt with, and the two reactions were in agreement.

Case 3 (fig. 6).—B., a fireman, aged 41, presented himself, July 24, 1920, complaining of weakness and swelling of the left ankle, tight feeling about the waist, and numbness in the feet, ankles, finger tips and left leg for a period of six weeks. He found it difficult to walk in the dark, and it had become

necessary for him to refrain from climbing fire ladders. There was a history of syphilitic infection in 1900, the treatment for which had consisted of mercury by mouth for a period of six months. The neurologic examination revealed a positive Romberg sign, ataxia of the upper and lower extremities, diminished deep reflexes in the upper extremities and absence of the deep reflexes in the lower extremities. There was impairment of touch discrimination and deep sensation in the lower extremities. The pupils were equal but irregular. The reaction to light was greatly diminished in the left eye but normal in the right.

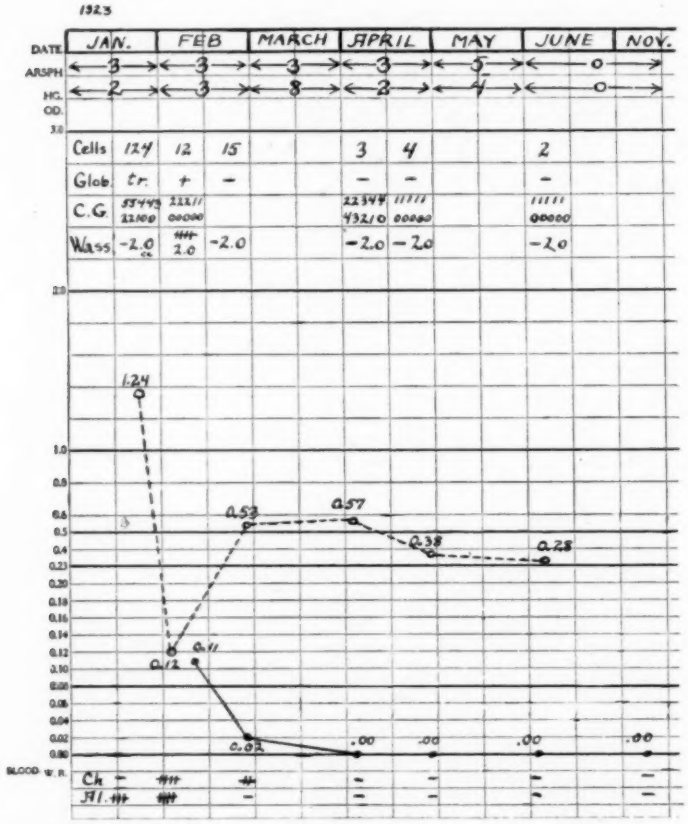

Fig. 7.—Comparison of Vernes and Wassermann reactions in patient H. The diagnoses were: etiologic — syphilis; anatomic pathologic — ectodermal; clinical — tabes dorsalis.

Treatment from June 24, 1920, to December, 1922, consisted of ten intravenous injections of arsphenamine (4 Gm.), thirty-one of silver arsphenamine (5.5 Gm.), forty-three intramuscular injections of mercury (59 grains), and twenty-three intraspinal injections. The subsequent treatment and laboratory observations in the blood and spinal fluid are indicated on figure 6. It will be noted that the Vernes readings in the spinal fluid in June, July and September, 1925, and March, 1926, indicated that the infection was arrested. The Wassermann reaction in the spinal fluid was positive on each of these occasions. Both tests remained positive in the blood during the whole period from June, 1922, to

March, 1926. The ataxia improved sufficiently to enable the patient to resume his occupation as a fireman, which required the climbing of ladders and scaling of buildings. The girdle sensation and dysesthesia disappeared. In this case the Vernes results in the spinal fluid indicated a steady regression of the syphilitic infection, and the clinical improvement justified this assumption.

CASE 4 (fig. 7).—H., a man, aged 46, without a history of any previous syphilitic infection, presented himself in January, 1923, complaining of difficulty in walking, cramp-like pains in the muscles of the thigh and ptosis of the left eyelid. The latter had been present on a previous occasion in 1919, following an attack of influenza, which was considered the cause at that time. It entirely cleared up in one week under the administration of potassium iodide, but recurred in December, 1922. Neurologic examination revealed an ataxic gait, positive Romberg sign, absence of deep reflexes in the upper and lower extremities, and Argyll Robertson pupils. The blood Wassermann reaction was negative with a cholesterinized antigen—water bath incubation—and three plus with an alcoholic antigen—icebox incubation. The spinal fluid examination revealed 124 cells; a trace of globulin, colloidal gold curve 5544322100, negative Wassermann reaction with 2 cc. and a Vernes photometric reading of 1.24. The Wassermann reaction was positive in the spinal fluid on only one occasion, and at the same time the Vernes result was lower than at any previous or subsequent examination. From January to April, 1923, pains of a radicular nature persisted, indicating pathologic activity. The cell count was also confirmatory of an inflammatory reaction. Four of the five examinations made of the spinal fluid during this period showed negative Wassermann reactions and positive Vernes readings. The Vernes results were more consistent with the clinical status than the Wassermann results.

SUMMARY

The following conclusions are drawn:

1. In sixty-two cases of neurosyphilis in which the Vernes and Wassermann tests were compared on repeated examinations of the spinal fluid, there was disagreement in three cases, or 4.8 per cent. The Vernes results conformed to the clinical status of these cases fully as well as, if not better than, the Wassermann.

2. In ninety-one cases of neurosyphilis, the two tests were compared on only one examination of the spinal fluid from each case. There was disagreement in fourteen cases, or 15.4 per cent. In correlating these results with the clinical aspects there was little choice between the two tests. The Vernes reaction appeared to conform better to the clinical aspects in eight cases and the Wassermann results better in six cases.

3. When only one examination of the spinal fluid can be made, the Vernes reaction furnishes limited information because a low reading does not necessarily exclude the possibility of neurosyphilis, and a subsequent examination might show such a rise in the optic density as to establish the presence of neurosyphilis. The same might be true of one negative Wassermann result, but the latter reaction does not show the variations in results from time to time.

4. In asymptomatic neurosyphilis or in symptomatic cases that are clinically stationary but with persistently positive Wassermann reactions in the spinal fluid, the Vernes test may furnish additional information of value in determining the therapeutic course to be pursued.

5. In cases in which the Wassermann reaction is negative but in which there is clinical evidence of pathologic activity, the Vernes test furnishes an additional laboratory method that may assist in clarifying the clinicopathologic concept of the case.

6. As an index of the character of the syphilitic infection, whether progressive, stationary or regressive, and as an index of the efficiency of therapy, the test recommends itself strongly, as it appears to measure the virulence or infectivity of the spirochetal strain in a manner that is not possible with the other tests.

METHOD FOR DEMONSTRATION OF SPIROCHAETA PALLIDA IN SINGLE MICROSCOPIC SECTIONS *

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In the study of the pathology of infectious diseases, no known organism has offered more resistance to the possibilities of constant demonstration than *Spirochaeta pallida*. In the laboratory of general pathology, the histologic diagnosis of syphilitic lesions could not always be supplemented with the staining of the organism. Furthermore, in lesions regarded as "suspicious of syphilis" a positive differential diagnosis could not be made on the basis of a negative result with the staining methods that have appeared from time to time. Even with the old Levaditi method for total blocks, the clinical and anatomicohistologic diagnosis of syphilis was often more significant than the absence of spirochetes by doubtful staining methods. Manouélian's modification of the Levaditi procedure made the technic more reliable, but such methods utilized the total blocks, often leaving no tissue available for ordinary histologic stains in cases of biopsy.

When Noguchi showed that spirochetes were present in the brain cortex of general paralysis, he immediately met a difficulty that had not presented itself before, namely, that nerve fibers absorb the silver impregnation in much the same degree as the spirochete. To Jahnel,[1] the neuropathologist owes the first noteworthy advance in the elimination of this complication. Through the use of uranium salts in aqueous solution, it became possible to prevent the impregnation of nerve fibers. With this contribution, he presented a method for use with microscopic sections of nervous tissue. Silver impregnation was made possible in such preparations through the principles of colloidal chemistry, as discovered by Liesegang,[2] with reference to diffusion in jelly-like media. Other authors, notably Steiner,[3] Krantz,[4] Warthin and Starry[5] and

* From the State Psychopathic Hospital, Laboratory of Neuro-Pathology.

1. Jahnel, F.: Studien über progressive Paralyse, Arch. f. Psychiat. **57**:847, 1917.

2. Liesegang, R. E.: Beiträge zu einer Kolloidchemie des Lebens. Steinkopff, Dresden, 1909.

3. Steiner, G.: Ueber eine neue Spirochäten Darstellung in Gefrierschnitt., München. med. Wchnschr. **69**:121 (Jan. 27) 1922.

4. Krantz, W.: Eine einfache Methode zur Darstellung der Spirochaete pallida in Schnittpräparaten, München. med. Wchnschr. **71**:608 (May 9) 1924.

5. Warthin, A. S., and Starry, A. C.: Second and Improved Method for the Demonstration of Spirochaeta Pallida in the Tissues, J. A. M. A. **76**:234 (Jan. 22) 1921.

Horálek,[6] have reported methods for single sections, involving manipulations based on the same idea. Jahnel regarded such preparations as useful for the study of spirochete morphology and for histologic reactions to their presence, as viewed in serial sections of brain tissue. With his technic he has contributed greatly to the knowledge of spirochetosis in general paralysis.

Stimulated by Jahnel's observations, I have carried out similar investigations. These have led to the development of a reliable technic for the demonstration of the spirochetes in single sections. The method here presented has been used in this laboratory for the past two years. Recently, it has been simplified and also made applicable to tissues other than those of the central nervous system. It has been found more satisfactory than any other method known, on account of its reliability, simplicity and time-saving features.

THE PRINCIPLES OF THE METHOD

1. After fixation and cutting, place the preparations in a 1 per cent solution of uranium nitrate in 70 per cent alcohol at 55 C. for one-half hour.

2. Wash for a moment in distilled water.

3. Pass the sections through 96 per cent alcohol, U. S. P.

4. Handling the sections individually, place them in an absolute-alcoholic solution of gum mastic (10 per cent) long enough to allow thorough infiltration—about thirty seconds. Immerse sections for an instant in 96 per cent alcohol.

5. Transfer them to distilled water.

6. Silver for from one to six hours at 55 C. in 1 per cent aqueous silver nitrate solution, carrying out this procedure without exposure to light.

7. Wash for a moment in water.

8. Develop in the following reducing solution for from five to fifteen minutes:

Hydrochinone	1.5	Gm.
Sodium Sulphite	0.25	Gm.
Neutral Solution of Formaldehyde[7]	10.0	cc.
Acetone	10.0	cc.
Pyridine	10.0	cc.
Water, to make	90.0	cc.

Mix and dissolve these and then add 10 cc. of 10 per cent absolute-alcoholic mastic solution to make the mixture "milky."

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6. Horálek, F.: Einfache Methoden zur Färbung von Syphilis Spirochaeten und Bakterien in Schnittpräparaten, Deutsche med. Wchnschr. **50**:988 (July 18) 1924.

7. Merck's neutral "Blue Label" 40 per cent solution of formaldehyde.

9. Wash for a moment in distilled water.

10. Dissolve out the mastic and dehydrate by transferring the sections to 96 per cent alcohol, U. S. P., and then to acetone. Clear in xylene and mount in Canada balsam.

The distilled water must be free from chlorides and all glassware must be likewise clean. Frozen and celloidin sections are handled with glass hooks and cover-glasses with ordinary forceps.

TECHNICAL PROCEDURES

Frozen sections of brain tissue or of the parenchymatous organs are cut from 10 to 15 microns thick after fixation in 10 per cent solution

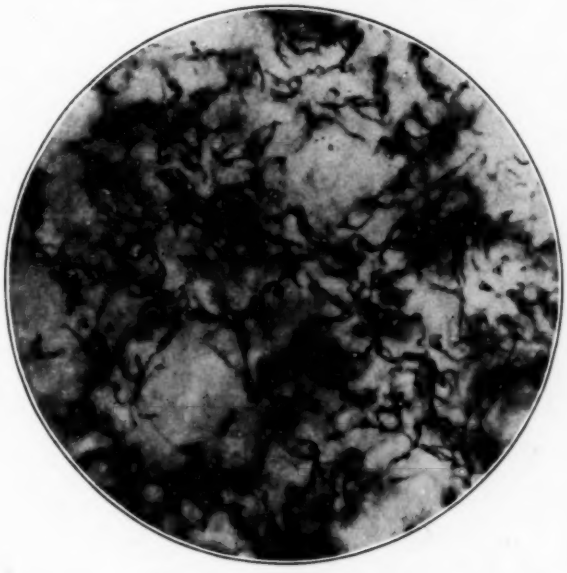

Fig. 1.—Spirochaeta pallida in the frontal cortex from a case of general paralysis. Solution of formaldehyde fixation, frozen section, 15 microns thick made twenty-four hours after autopsy. × 667.

of formaldehyde for at least twelve hours for small blocks or slabs of tissue. Fresh material, that is, the more recently fixed, affords excellent results. Figure 1 shows the organisms from a case of general paralysis in a section of cortex prepared less than twenty-four hours after autopsy. In the same case I was able to demonstrate them six hours post mortem. Excellent results are obtained in tissues preserved for more than ten years. The age of such solution of formaldehyde and the acidity of the ordinary commercial variety do not seem to affect the process of silver impregnation, as has so often been emphasized.

Celloidin sections, from 10 to 15 microns thick, from recent or old formaldehyde or alcohol fixed material, give equally good results. With

these, the celloidin is dissolved out in acetone before one proceeds with the first step. They are then manipulated as described for frozen sections.

Paraffin sections, from 10 to 15 microns thick, after fixation in solution of formaldehyde or alcohol, may be affixed to the cover-glass or to slides with albumin fixative and dried. After removing the paraffin with xylene and passing the sections through absolute alcohol, one proceeds with step 1. With cover-glass and slide preparations it is possible to stain the smallest pieces of tissue, such as secondary papules, and the same manipulations can be applied to the largest type of section that can possibly be cut with the microtome. Paraffin sections cut for routine diagnosis and floated onto a glass plate with molasses or dextrin fixative

Fig. 2.—Spirochaeta pallida in and about a cortical blood vessel from the same case as figure 1. Alcohol fixation, celloidin section 15 microns thick. × 333.

for the preparation of a celloidin sheet may be treated as described above for celloidin sections.

Smears from the primary lesion or made from tissue emulsion by rubbing together small bits of the organ, especially general paralytic cortex, with a drop of physiologic sodium chloride solution, are fixed for one hour in solution of formaldehyde-alcohol (10: 90 of 96 per cent) and then introduced into step 1 after they are rinsed in distilled water.

Uranium nitrate in alcoholic solution inhibits the absorption of silver by nerve, collagen and elastic fibers, making the field clear of elements that are apt to confuse the examiner. If the stainable fibers persist, one

may experiment with stronger concentrations of uranium. I have used a 5 per cent solution to advantage. Similarly, a solution weaker than 1 per cent may be required in older tissues. The alcohol in this uranium solution and the 96 per cent following it serve to after-harden and to prepare for receiving the alcohol-soluble gum mastic.

In the mastic solution, the sections are left about a half minute to allow for infiltration. They are best handled separately during this step. The gum so affects the thinness of a section as to make it physically comparable to a total block. (The mastic solution is prepared by dissolving 10 Gm. of gum mastic in 100 cc. of absolute alcohol. About three days is required for complete solution. Then filtration is made through three-fold filter.)

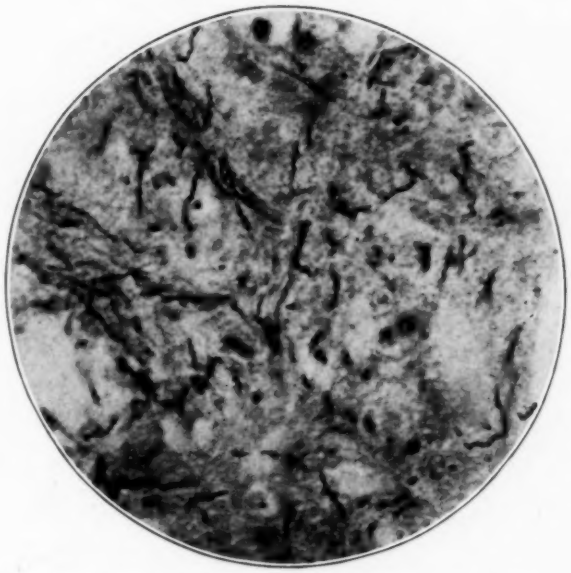

Fig. 3.—Spirochaeta pallida in the connective tissue of a genital chancre. Solution of formaldehyde fixation, paraffin section 10 microns thick, cover-glass preparation. × 667.

In water the gum mastic readily precipitates as gummy strings, thus "padding" the sections. After this the momentary immersion in 96 per cent alcohol allows the proper amount of mastic to be retained within the sections. The sections may be collected at this point or placed singly into the silver solution without stopping to gather them together in one dish. Thorough precipitation of the mastic occurs in about a half minute.

Silver nitrate in concentration of 1 per cent is chosen because heavier solutions tend automatically to reduce at 55 C. After the preparations are exposed to this solution for one hour, trials for the success or degree

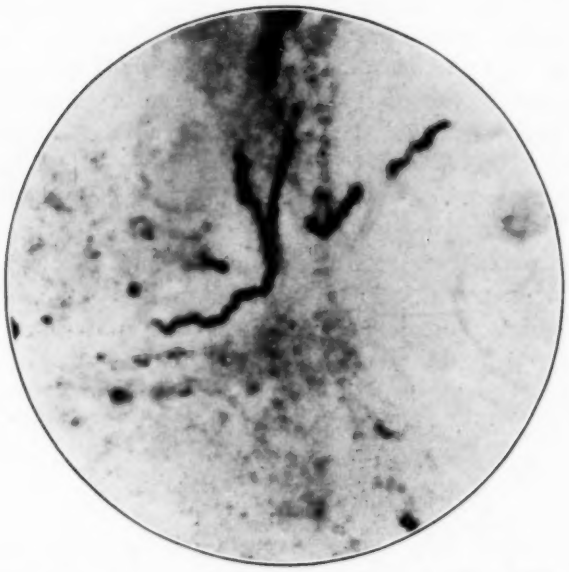

Fig. 4.—The same organism shown in figure 3 at region of "4 o'clock on the dial." ×2000.

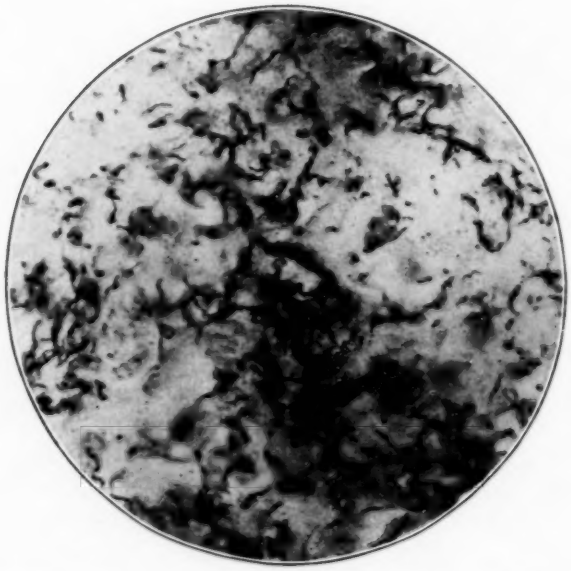

Fig. 5.—Spirochaeta pallida in fetal liver. Old material, preserved in solution of formaldehyde, paraffin section 15 microns thick, cover-glass preparation. ×667.

of impregnation can be begun by washing and reducing. Usually three hours in silver suffices. If the technician wishes to suit the method to his convenience he may allow the silvering to continue over night, but this must occur at 37 C. on account of the automatic reduction mentioned. In my experience, older tissues may require much longer and milder impregnation and the use of silver nitrate in concentration of 1.5 per cent. After silvering, wash the tissues for a moment in distilled water to remove the excess. If the heat has clumped the frozen sections

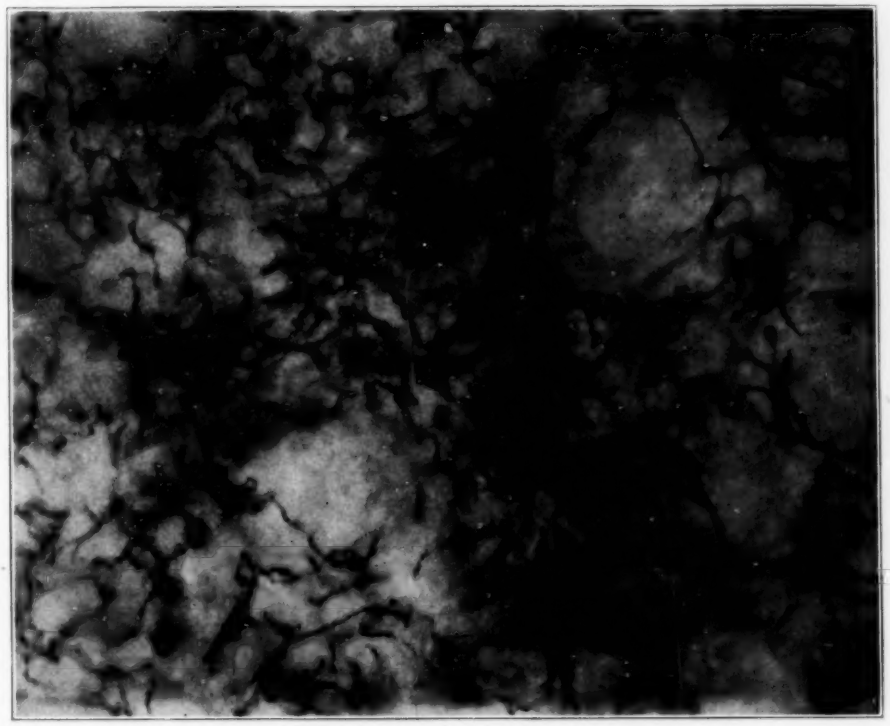

Fig. 6.—Showing typical morphology of Spirochaeta pallida by the author's method. The even quality of the impregnation with absence of "dirty" silver precipitates and of nerve-fibers should be noted. General paralysis. × 1333.

into a bunch by making the mastic gummy and wrinkling them, this does not spoil the results in any way. It is better to handle about twelve sections in one dish of 100 cc. silver nitrate solution.

Reduction in the developer usually occurs immediately. The acetone and pyridin components dissolve the gum mastic in the sections, but the presence of the gum in the developer, as added, permits the physicochemical process to obtain. Individual tissues have slight variations, thicker and older ones requiring the maximum time in this reagent, which is so formulated as not to allow overdevelopment and to permit

use for many sections. The solution turns creamy yellow to orange on standing, but this is one of its virtues, for the color is transferred to the section much as an acidophilic dye, and in fresher tissues it makes a good nuclear stain. The sections may be developed individually, or several can be allowed to develop at once. Frozen sections should be flattened out to insure even tone of reduction.

The final rinsing in water serves to remove the developer. The 96 per cent alcohol and the acetone dissolve out the mastic and dehydrate. The reducing quality of acetone is an advantage here. The xylene and mounting in balsam follow as usual.

In all the procedures, the final result produces sections that are of a beautiful golden yellow to golden brown according to their thickness. The spirochetes are brown to black in proportion to the heaviness of impregnation, although it is interesting to note that the more intense the spirochetosis and the thicker the section the blacker will be the color of the organisms. The best results with photomicrography occur with the use of a red filter for the demonstration of the organism itself, and with a light blue one for detail in the histologic relations.

With the procedures described in the foregoing, I have been able to find the spirochete more easily and constantly than with any other method. The whole procedure requires as little time and actual care as an ordinary substantive stain. In one evening I was able to prepare a set of fifty specimens for use in class in the laboratory course. After learning the principles, the technician may vary the method to his own convenience. This we have done, and it has enabled us to study in serial sections the spirochetosis of general paralysis in coordination with histologic changes. The results of such studies will be reported at a later date.

CEREBRAL CIRCULATION *

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The work of Krogh [1] on the anatomy and physiology of the capillaries furnished a new method of approach for the study of the vascular system. His work is so well known that it is unnecessary to dwell on it further than to state that he observed the circulation in superficial vessels through a binocular microscope. Microscopic study of the capillaries in most tissues, however, offers many difficulties, one of which is the lack of contrast, the vessels being of a color similar to that of their surroundings. It occurred to us that the brain would be the best place to study the capillaries, as here the contrast would be greatest and the observations more accurate and reliable.

Our work is being carried out along the following lines: (1) determination, by direct microscopic observation, whether the cerebral vessels are affected by stimuli which cause vasomotor effects in vessels of other portions of the body; (2) investigation of the effects of drugs on the vascular system of the brain; (3) study of the causes and effects of thrombosis and embolism; (4) observation of the phenomenon of inflammation, and the evaluation of the effect of treatment directed to its alleviation.

In the present report we shall not discuss this entire field, but we shall make a short outline of the methods used, and give the results of some of the experimental studies.

METHODS OF STUDY

The animals studied were mainly cats, though rabbits and frogs were also used.

Ether administered by the intratracheal route or solutions of chloral hydrate or urethane given subcutaneously were the anesthetics used in most instances.

Intratracheal anesthesia is preferable, because when the movements of respiration are diminished, the corresponding excursions of the brain are lessened, and a steadier image of its surface is thus obtained. This is especially necessary when the higher magnifications are used.

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* Read at the Fifty-Second Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1926.

1. Krogh, August: The Anatomy and Physiology of Capillaries, New Haven, Conn., Yale University Press, 1922.

The cortex of the brain was always exposed through a large opening. If the observations are made through a small aperture there is an artificial movement and pulsation in the vessels caused by the intermittent pressure of the bulging brain against the margins of the bone.

The microscope used is a Bausch and Lomb binocular, which gives stereoscopic vision. Magnifications of from 19 to 176 diameters were obtained, though a magnification of 80 diameters was found most satisfactory. With this power the circulation could be seen in the small capillaries under ordinary conditions.

The movement of the circulation can be observed only if the rate is comparatively slow. For this reason, it cannot be seen in the larger vessels under normal conditions, but can be perceived in the capillaries, in the smaller arterioles and in the small and medium-sized veins. The normal circulatory rate in cats seems to be slower than that in rabbits, and on this account minor changes in the rapidity of the circulation are more easily detected.

APPEARANCE OF THE CEREBRAL CORTEX UNDER NORMAL CONDITIONS

The vascularity of the cortex varies exceedingly in individual animals. It is much greater in cats than in rabbits. Some rabbits present hardly any visible capillaries and only a few scattered arterioles and venules. This appearance is also frequently seen in frogs. In these animals one or two vessels may be all that can be seen over the entire surface of the brain. While there is a good deal of individual variation in the number of visible blood channels in the cortex of cats, many capillaries are always present and are for the most part larger than what may be called "one-cell" capillaries. These are the smallest vessels, and do not admit of more than a single file of corpuscles. The larger veins and arteries occur together, and, in the main, course along the sulci. In some instances and in some situations the veins are more superficial, and in others the arteries are in this position. We have not been able to discern any constancy of arrangement. The smallest arteries and venules, in most instances, do not remain together, but cross the gyri in irregular fashion. The veins are easily distinguished from the arteries by their color. Venous anastomoses are of frequent occurrence; the direction of the current in these connecting vessels varies from time to time, occurring sometimes in one direction and at other times in the reverse direction. Pulsatory movement is seen at times in the larger arteries, but variations in the rate of the current are not discernible.

Direct Stimulation of Cerebral Vessels.—The blood vessels of the cortex are beneath the arachnoid, which in this situation is simply the out layer of the pia. It is only slightly separated from the lower layers

over the vertex of the gyri, where there is only sufficient space for the blood vessels and a thin layer of cerebrospinal fluid. It bridges over the sulci superficially, without dipping into the folds, and in this position is separated from the inner layer of the pia by a considerable space which is filled with cerebrospinal fluid. In this region the membrane is thickened and strengthened by strands of glistening fibrous tissue.

The blood vessels of the cortex are not only covered by the arachnoid and by the cerebrospinal fluid, but are also surrounded by a lymph space. This perivascular lymph space is not visible under ordinary conditions, but is plainly seen in inflammations of the cortex or when a portion of a vessel is strongly contracted by the faradic current.

The arachnoid cannot be removed without injury to the delicate vessels which are connected to it by fine trabeculae. The application of the solutions used in this experimental work was therefore made to the outer surface of the arachnoid over the vertex of the gyri, but it is easily conceivable that the negative effects in some instances may be due to lack of penetration, the solutions not coming in actual contact with the walls of the vessel.

Mechanical Stimulation.—Compression or rubbing the arteries is said by Florey [2] to result in a contraction localized to the point stimulated. In repeated experiments with fine glass rods, horse hair, fine wire, hypodermic needles and other objects, we have never seen the slightest evidence of any effect on the caliber of the arteries, veins or capillaries, after the compression is withdrawn. In rabbits, stroking the vessels with a cotton swab makes the perivascular lymph space visible, but does not alter the size of the vessels themselves.

Electrical Stimulation.—Stimulation of the arterioles with a faradic current, using a bipolar electrode, causes a sharp contraction at the point stimulated. The vessel contracts to one half or one third its original diameter over a length equal to six or eight times the original diameter. After this contraction it requires about ten minutes for the vessel to resume its original appearance. Some of the capillaries show similar responses, but the veins are unaffected.

Direct Application of Epinephrine.—Direct application of epinephrine in strengths varying from 1 : 1,000 to 1 : 10,000 does not usually have any effect in the cat or in the rabbit. In a few instances definite dilatation was observed. The enlargement may be symmetrical, or the vessels may present a slightly varicose appearance. In the frog, dilatation is regularly observed.

Ninety-five per cent alcohol applied to the cortex rapidly penetrates the capillaries, causing coagulation and thrombosis with complete cessa-

2. Florey, Howard: Microscopical Observations on the Circulation of the Blood in the Cerebral Cortex, Brain **48**:43, 1925.

tion of circulation. In some of the slightly larger vessels thrombi are formed, which may narrow the lumen of the vessel without stopping the circulation. In other instances these white thrombi may break away from the vessel wall and be carried away in the blood stream. The larger arterioles and venules show little change except that small white thrombi may be formed on the vessel walls. They are at first small and look like minute white specks, but they rapidly increase in size, and are eventually dislodged and borne away by the circulation.

A drop of paraldehyde applied to the cortex causes an immediate coagulation of the cells of the pia-arachnoid and of the blood in the smaller vessels directly beneath it. In the largest vessels, in which the circulation is moving swiftly, obliterating thrombosis does not occur. On the walls of these vessels small white thrombi were formed and swept away when they had grown to sufficient size to project moderately into the lumen of the vessel.

Similar phenomena are seen when dilute hydrochloric acid, tincture of iodine or other irritating substances are placed on the cortex.

EFFECTS OF INTRAVENOUS INJECTIONS ON CEREBRAL CIRCULATION

Epinephrine Hydrochloride.—Injection of from 1 to 3 cc. of 1 : 1,000 epinephrine hydrochloride solution did not cause any perceptible change in the size of any of the cerebral vessels in cats and rabbits. There is an acceleration of the circulation, and after doses of from 3 to 5 cc., spontaneous hemorrhages are seen. In frogs, hypodermic injections of epinephrine hydrochloride cause dilatation of the cerebral arterioles, which is at first so great that there is momentary cessation of the circulation. In a few moments it is resumed again, and in some of the connecting vessels the direction of flow is reversed.

Pituitary Extract.—We have never observed any effect after the injection of 1 or 2 cc. of pituitary extract, either under normal conditions or when the arterioles are contracted by the faradic current or dilated by histamine.

Paraldehyde.—Intravenous injection of 0.5 to 1 cc. of paraldehyde produces a pronounced slowing of the circulation, so that the flow is visible in the larger as well as the smaller vessels. There is also a gradual accumulation of fluid beneath the pia-arachnoid, which is evidenced by an increase in the depth of the space between the surface and the cortex. The circulation in the capillaries becomes slow, and in some vessels it ceases altogether. If 5 cc. or more is injected into the femoral vein, there are irregular, momentary annular contractions of the arterioles, and fluid accumulates rapidly in the pia-arachnoid spaces. This edema forms so rapidly that it produces an irregular elevation of the

arachnoid and a rupture of some of the fine intervening trabeculae. The circulation rapidly ceases in many of the capillaries and smaller vessels, and is markedly slowed in those of larger caliber. Within three or four minutes the circulation stops entirely.

Hypertonic Solutions.—Intravenous injection of 10 cc. of 25 per cent sodium chloride solution or 100 per cent dextrose solution did not produce visible alteration in the blood vessels. There is absorption of some of the spinal fluid, so that the surface of the arachnoid is not flat, but is irregular and outlines the vessels under it. Rabbits inoculated with herpetic virus show pronounced dilatation of the perivascular lymph spaces. Under these conditions, the administration of hypertonic solutions causes shrinkage and disappearance of these spaces, due undoubtedly to the passage of the fluid distending these channels into the blood vessels, which they surround.

India Ink.—In order to trace the course of minute foreign bodies in the blood stream a solution of India ink was used. The individual particles of carbon in this suspension are smaller than bacteria, being only about 200 microns in diameter.

After intravenous injection of 3 cc. of a mixture of equal parts of India ink and normal saline solution, there is an immediate darkening of the color of the blood. Darkening appears before all of the ink has been injected, and it continues for ten or fifteen seconds after the injection. As the color is returning to normal some black particles are observed suspended in the circulation. The extremely rapid removal from the circulating blood of this foreign matter is astonishing. For the next three or four minutes there is little to be seen except an occasional black particle floating in the circulation. At the expiration of this time, more minute black particles are seen. Some of these may adhere to the vessel walls, especially in the veins in which the circulation is slower. Soon these particles have increased in size and occlude some of the capillaries. In a few minutes large masses, which seem to be made up of particles of ink held together by some transparent substance, are seen passing through the arterioles. When a large mass encounters a fork in the vessel it lodges and blocks the circulation. This obstruction may be permanent, or in a few moments small portions of the embolus may be broken off and washed away down one or the other of the branches. In this way the entire mass may be broken up and removed, and the circulation may be reestablished. If 5 cc. or more of the ink mixture is injected at one time the animal will not live more than about fifteen minutes, and by the time death occurs many of the vessels of the cortex are occluded by these emboli. Examination of the emboli shows that they are made up of carbon particles and blood platelets. The platelets have apparently been the agglomerating agent.

Tap Water.—Tap water, at a temperature of 90 F., given slowly by infusion into the femoral vein of a cat weighing 2,200 Gm., caused the following phenomena: No effect was observed until 40 cc. had entered, but at that time the respiration suddenly ceased and was not resumed again; the action of the heart was unaffected. When 60 cc. had been given, the circulation was markedly slowed, so that the corpuscles could be observed in the larger arteries. On close examination the corpuscles seemed deformed. When 68 cc. had been injected, small white thrombi commenced to form on the walls of the vessels and, breaking away, were carried along in the blood stream. After the administration of 75 cc., the blood appeared definitely laked, and normally formed cells could not be seen. When 80 cc. had been administered, the circulation ceased.

NEUROPSYCHIATRY IN CHINA

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An editorial in the *China Medical Journal,* in 1889, stated that nervous and mental diseases were remarkably infrequent in China; unfortunately this statement is still being made repeatedly by those who should know better. That such diseases form only a small part of the total number of cases that find their way to China mission hospitals is doubtless true, but this is also the case in large general hospitals in any part of the world. Nevertheless, a perusal of the yearly reports from the many mission hospitals and of questionnaires sent to about 350 physicians throughout China stimulate the estimate that there are at least 3,120,000 cases of nervous and mental diseases in China, and that one of every 126 sick persons is a neuropsychiatric case. From the figures given, it is concluded that at least 43 per cent of these cases are purely psychiatric; this shows that there are 1,341,600 insane or psychopathic persons in China, which undoubtedly is a great underestimate. It is usually thought that the average Chinese leads an easy-going, quiet life, and that therefore the many evil effects of the high-pressure life of Occidental cities are scarcely to be expected in China. If such is the case, this makes the investigation of nervous and mental diseases in China all the more important. At least 100 physicians in various parts of the eighteen provinces of China have emphasized in their answers to the questionnaires that the need for specialization in this subject and the establishment of neuropsychiatric hospitals in advantageous places is imperative, although at present not any private neuropsychiatrists in China devote their whole attention to this subject. Only six physicians in this teeming nation express more than passing interest in the important problem.

To date there are not any national government hospitals for the insane in China. In Peking, North China, there is a place that is sometimes called a hospital for the insane, but it is nothing more than a prison in which psychopathic persons are confined and sometimes put in chains. This so-called hospital is under the charge of a Chinese doctor who received his training in Japan. Also, in Canton, South China, about 1,200 miles away, there is a Municipal Insane Asylum in which 200 or 300 patients are "herded" together. Christian missions are doing a little more for these unfortunates. There is a small Roman Catholic hospital in Shanghai for twenty patients, and two or three hospitals for Protestant missions set aside a ward for mental cases. So far, the only large institution devoted wholly to the care of psychiatric

cases and conducted with a scientific outlook is the John G. Kerr Hospital in Canton, which has a capacity of a few more than 700 patients; it has always been reported on favorably, although the unsettled condition of the country has recently caused it to close. Soochow boasts of a small "Insane Asylum" outside the city, which is under the charge of the Presbyterian Mission; this institution has been reported on unfavorably, because of its lack of scientific care. The Peking Union Medical College does not provide any separate accommodation for mental cases, although it reports the treatment in a goodly number of psychopathic cases, both Chinese and foreign. For the care of foreign psychopathic patients, Shanghai offers a limited ward for a few acute cases; Hongkong has a government lunatic asylum; and the Hankow International Hospital has one room for mental cases. Unfortunately, these places all have inadequate facilities for proper psychiatric care.

The earliest recorded attempt to treat mental diseases by the Chinese dates back to 1122 B. C., at which time magic, sorcery or mesmerism were practiced much as they are today. The first record of the treatment of psychiatric cases by foreigners in China is shown in Dr. Dudgeon's Peking Hospital Report of 1868, in which he states "many cases of unsoundness of mind came under treatment, but there are very few of a violent character."

Patients with mental defects constitute a helpless class in China. If caught on the street doing anything unusual, they are arrested and thrown into prison as if they were criminals. If they are harmless and wander into the streets, they are mocked and laughed at, and are often stoned. Their families usually treat them as strangers and confine them alone in a dark room. If they tear their clothes or are otherwise destructive their closest relatives sometimes disown them, although occasionally there may be true affection shown by a family for a psychotic member, especially a son. Most patients are kept chained at home and are not allowed to go abroad, with the result that few people know about them. Restrictions are not put on them by the government. As long as they are not found stealing or suspected of it, or if they do nothing violent, they may go about at will as far as the government is concerned.

In China the head of the family is usually held responsible for the injurious acts of any of its members, which accounts for the fact that psychopathic patients who are dangerous are rigidly confined either at home or in a monastery, and the disposition to treat them cruelly is enhanced by the fear and dislike inspired by the belief that a psychosis is due to possession by evil spirits. Several cases have been reported, in which the mentally unbalanced person was built into a small cell-house, without any door and only a small window to admit food. Thus the circumstances are usually such as absolutely to preclude the patient's

recovery. Food, clothing, bedding and the necessities of life are most sparingly supplied. Baths are infrequently, if ever, given. A mentally defective person is considered by his family as being lost; as long as he lives he is a burden to all concerned. The treatment is probably not always intentionally cruel, but is rather that of neglect. Many of these patients would undoubtedly recover or improve under better treatment, for in a series of about 350 Chinese patients given proper treatment, 26.2 per cent were discharged as cured and 45.2 per cent as improved.

Except for a few educated persons, the Chinese know nothing of the real nature of a psychosis, and the friends of a patient with a violent mental disease do not have recourse to any other efficient plan of management than the use of chains. Because the father potentially has the "right of life and death" over the members of his family, he may choose the shortest and most effectual method of treating a psychopathic member of his household and dispatch him without delay. It is probably seldom that parents themselves dispose of such sons or daughters by killing them, although if these children are left in the care of strangers undoubtedly they sometimes meet such an end.

A case is reported of an insane woman in Canton who had been chained about the neck to a heavy stone for fifteen years. The chains used for men are sometimes heavy, and the patient is often chained neck, hands and feet. Many bear the marks of the whippings or poundings they have received or of fetters that have been on the extremities. Two cases are reported, one of a slave girl, the other of an old man, with thumbs badly burned. This mode of torture is inflicted by placing on the thumb nails a little piece of wicking soaked in oil or kerosene, which is then lighted and kept in place. The object of this procedure is to determine whether the victim is merely obstinate and disobedient or is really "insane." It is also used sometimes as a remedy "to drive out the devil" of insanity. In one part of China the psychotic individual is taken into the country and a large stone is placed on his chest. If this does not crush out the devil, the patient is usually allowed to starve to death with the stone on top of him.

It is the almost universal belief among the Chinese that psychotic persons are under the influence of evil spirits or the devil. This belief is not surprising when it is realized that twenty-four of the missionary physicians questioned answered that they had seen cases that they considered were of demoniacal possession. Another belief widely held by the Chinese is that the immediate cause of a psychosis is the presence of mucus choking up the internal organs within the chest (which may mean the heart, lungs or even the stomach, so indefinite is the knowledge of what really exists or the location of each). Some educated Chinese

place the seat of the disease in the brain, and say that it is brain sickness or a diseased mind, caused by sorrow, excitement, trouble, anxiety, fear, anger, or repression, or perhaps just malingering. There are other beliefs: that the patient has been led astray by bad spirits; is possessed by the "Fox"; that it is a heart disease, a loss of soul, or a wind disease; or that it may be the result of heart phlegm, sputum or fire in the system; that the patient's ancestors have not been interned according to rule, or that the patient is just "Fung-la," or crazy.

Native psychiatrists are not to be found in China outside the two or three institutions already mentioned. Exorcism is a common practice throughout the country, and according to reports some Christians even can be secured to attempt this "psychotherapy." Since native doctors are usually "alchemists" or cure-alls, it is not surprising that they claim to be able to cure mental diseases, or at least to cure the patient himself, for in most cases they manage to put the patient out of his misery. One remedy used by them is "tung-yau," commonly known as "wood oil." It is administered in the form of little cakes containing the oil. Repeated doses of this oil cause violent and distressing vomiting, which occasionally proves fatal. In using this treatment the aim of the native doctor is apparently to make his patient throw off his insanity. Croton oil cakes are also used to cause emesis and purging in order to "get rid of the phlegm." Hyoscyamus or henbane is one remedy sometimes used by the Chinese, which fortunately in some cases may be beneficial. This plant is indigenous to many parts of China. A tea is made from it and administered in bad cases of mania. Whether this is done without any foundation or belief in its value, or whether it has been found by experience to have a quieting effect in cases of mania, is not known. Another method of treatment is for a "witch doctor" to say incantations over the patient. These magic words are then written on paper, which is burned. The ashes are mixed with water and given to the patient to drink. Pills containing sulphur are sometimes given, and in cases of epilepsy a pill containing bezoar, croton bean, arsenic and cumabar is given. If the "soul is lost," it is gathered together by sweeping with a broom near where the patient took sick, while members of the family summon back the soul by calling the patient's name. Needling of various parts of the body also has been reported. In Manchuria, a common medicine for mental disease is an infusion of green beans and lycopodium. Beating and striking the patient with the hand is a common practice, intended either to awaken the insane mind or to drive out the devil. A hot piece of iron is sometimes used to burn the patient in spots, or a burning coal or wick is placed on the thumb nails. In some cases feces and urine are thrown on the patient, apparently to disgust him into a cure. All the methods

of sorcery are of course used by the priests, and occasionally the patient is taken to a temple to sleep before an idol in the hope that he will be cured by a dream revelation.

From the questionnaires, it was gathered that between 1 and 30 per cent of the general Chinese populace have psychopathic tendencies, or may be considered border-line cases. One physician in Chihli, a northern province, even went so far as to estimate the percentage at 75. Of course it is difficult for the average Occidental to form a fair gage of Chinese mentality, since Occidental and Oriental moral standards are so different. Nevertheless, underlying all, man's primitive mentality is the same, and in due time research will clarify Chinese psychology.

Reports from physicians throughout China who have cared for psychiatric patients show that the precipitation of mental illness in each patient, whether Chinese or foreign, was assigned to such causes as business anxiety, death of a child, death of mother, lack of attainment, disappointment in love, domestic troubles, excessive study, loss of property, political or war excitement resulting from politics or war, loneliness, sexual maladjustment, religious dominance, fear of spirits, force of circumstances, or inability to adjust to foreign environment.

Syphilis is rife throughout China, and as an exciting cause of psychopathologic conditions is undoubtedly as common as in either Europe or America. Alcohol is probably not used as much among the lower classes in China as in Europe, but there are a great many Chinese of all classes, especially in the middle and upper classes, who regularly take alcoholic drinks. Opium is again becoming widely used and is likely a contributory factor to psychopathologic conditions. The Chinese are universally infested with parasites, and a great many have ever-present foci of infection. The last two factors often have been found to be the exciting agents of a psychosis, and when a chronic abscess or a "mess of worms" is gotten rid of, the patient has regained a social mentality.

The third decade of life apparently is the crisis here as elsewhere, for of all the mentally ill coming under care in China, 40 per cent are between 20 and 30, 23 per cent between 30 and 40, 9 per cent between 40 and 50, 5 per cent over 50, and 23 per cent under 20 years of age. From the questionnaires, it was found that 48.9 per cent of Chinese patients with mental disease are men, and 51.1 per cent women, although of these, 65.4 per cent of the patients discharged were men and 34.6 per cent were women.

A study of the statistics clearly shows that the proportion of insanity is highest when there is the greatest congestion of population, and when the stresses incident to active competition are most severe. If the map of China is scrutinized, it will be found that the highest rate of mental illness is in Chihli, Shantung, Chekiang, Fukien and Kwangtung, with Hupeh, Anhwei, Kiangsu and Honan coming close to them. These

provinces all have a population of more than 250 persons per square mile, while Kansu, Yunnan and Kwangsi have less than 100 persons per square mile and also have the lowest rate of psychoses in China. It appears that psychopathologic disease increases in proportion as the stresses incident to the struggle for existence become mental stresses, such as is found in the big commercial cities: Canton, Shanghai, Peking and Hankow.

The places in China with a low insanity rate are settled by hardy farmers, who produce a strong human stock. It is different, however, in the great commercial cities mentioned, to which all the wandering, unsettled riffraff of the country have hastened in the hope of acquiring a so-called life of happiness and who yield to all the seductions of intemperance, with resultant increase in mental weakness. Likewise, along the Chinese coast, the off-scourings of other nations have drifted to these shores looking for that wealth they always think should be ready at hand, with the result that many of the psychoneurotic persons in the coast towns and treaty ports are of foreign birth.

From the questionnaires it was concluded that 43 per cent of the neuropsychiatric patients in China have psychoses and need institutional care. The remaining 57 per cent have border-line cases or episodic states, who, although not needing institutional care, need professional guidance. Among the cases coming under the attention of the physicians questioned, practically every type of mental disorder has been reported. A physician in Hupeh said that he had seen all kinds of cases "from the inspired idiot to the suppressed genius—from the wildly maniacal to the morosely suicidal." Averaging the reports, it was found that 27.7 per cent of the cases are of the manic-depressive type. The next largest group, 22.4 per cent, are the psychoses associated with organic disease or injury of the brain, the greatest number of these being epileptics. General paralysis, although reported by some authors as being absent or seldom seen in China because of the frequency of malaria, is given as the diagnosis in 12.1 per cent of the patients. Dementia praecox or schizophrenia is present in only 11.1 per cent. Patients with paranoia or paranoid states are often mistakenly classed as having dementia praecox; they really belong in a class by themselves, since they do not have intellectual impairment. This class of patients make up 7.5 per cent. Persons with some neurosis that need institutional care constitute 5.8 per cent; the presenile, senile and arteriosclerotic psychoses come next, with 5.5 per cent. Idiocy and imbecility do not appear to be common in China, for only 3.4 per cent of the total are of this type. The toxic psychoses, which include patients addicted to opium or alcohol, make up 2.2 per cent. The infection and exhaustion psychoses take a toll of 1.9 per cent, while the symptomatic psychoses,

which include patients suffering from pellagra, multiple sclerosis, chorea, uremia, etc., constitute only 0.4 per cent of the total neuropsychiatric cases reported.

The symptoms of Chinese patients are fundamentally the same as those of foreign patients, although the mode of exhibition is somewhat different. It has been claimed that the Chinese are able to bear, without flinching, a degree of pain from which the stoutest foreigner shrinks in terror; in actual fact, the Chinese have just as sensitive a psyche and are also subject to "nerves." Chinese soldiers are the class that should be able to meet pain with greatest fortitude, yet once they experience the pain, it is the rule rather than the exception for them to shrink from it; they beg to be given an anesthetic for even trivial operations, or flatly decline to be operated on at all.

A Chinese coolie can stretch himself on one or two hard boards and calmly go to sleep amid the noises of a busy street. This is not proof of the absence of nerves, for if the same coolie is near a wood at night, the rustling of the wind through the branches of the trees will make him sleepless owing to his superstitious fears. A foreigner visiting a Chinese outport town for the first time finds the native watchman with his gong an unpleasant interrupter of sleep, while the old resident slumbers on undisturbed. The ability to sleep amid noises is simply a matter of habit or training. The Chinese has a stolid aspect, is deliberate in speech and movement, and fails to appreciate the value of the golden moments as they pass, but this again is not a sign of a lack of "nerves."

The Chinese social system has its strong and its weak points, as has that of every other nation. It is about these points, particularly friction in the household economy, that much pathologic thinking gathers. Some of the points can be traced through the construction of the language. Polygamy and concubinage are common in China, and the struggles, the fears and the hates between wives and concubines and the cruel treatment that the latter generally receive are often an etiologic factor. Then there is the domineering Chinese mother-in-law and her treatment of her daughter-in-law and female dependents. Many pitiful cases of abuse under the system of child slavery are to be seen in China. Mere children are sold into households or brothels and some are so terrified that they become deranged, usually developing first a neurosis and later a psychosis.

Wherever there are men and women, betrothal and marriage relations form the subject of much thought and speculation. Delay, disappointment and abuse cause much trouble. To the average Occidental, the Chinese betrothal and marriage customs are unemotional, but such is far from being the case. With certain unstable natures, whether Chinese

or foreign, the mere approach of the time for consummating the marriage relation is sufficient to cause such morbid apprehension that they become psychically upset. Not infrequently, in both sexes, a peculiar antagonistic attitude may be found. Many Chinese are of homosexual character and say they are always troubled by the opposite sex.

In Chinese patients, the fear of devils and evil spirits takes the place of what is commonly known as "religious mania" in Occidentals. The usual Chinese, who is generally not a Christian, when suffering from a neuropsychosis or psychosis, does not think he has committed an unpardonable sin, but he fears the devils. He sees devils; he hears them; he feels them; he is possessed by them, and is tormented by them in every conceivable manner. This is, of course, the result of the inherent Chinese superstitions. It is supposed by the Chinese, as well as by other nationalities, that devils are prone to make peculiar noises at night in the upper loft or on the roof. In most parts of China it is imagined that spirits make noises like rats, ducks and other animals; that they cry at night, and call for money and food; that they are seen perched on the roofs, which may account for the typical roof decorations found on Chinese buildings; that they flit vaguely in and out of open doors and windows; that they are seen as black shadows on walls, and they are often said to be very large. Some of the female devils are said to be tiny, and some are headless; they frequent dark corners of the room and get under the bed; they have a tendency to pull the bedding and even the legs of the unfortunate patients. All of these hallucinations are apparently unconscious creations to bolster up the patient's potency.

Heavy responsibility suddenly thrust on a youth by the death of his father is a situation that frequently marks a break of the psychic balance. Especially is this true in China, where the social system makes the father the dominating power. The youth thus placed worries about the problems of life without any buffer between himself and the outside world. Patients of this type will often present the reverse character in their delusions; they feel capable of meeting the responsibility of the highest public office; they become magistrates, tupans, kings, emperors, presidents, and the like. All their fears of inefficiency are dispelled in their new mental world, and they become greatly extroverted.

The Chinese wife who has not borne a male child is haunted by the fear that her husband will take a concubine; her "reproach" is a real thing and weighs heavily on her mind. She may develop ideas of persecution and assert that her husband has concubines in other places. The loss of children, or the approach of the climacterium, is also the cause of much anxiety in Chinese patients.

The Chinese are a vast storehouse of psychic meanings, which accounts for them being known as "a puzzle," and objects for solution.

The trouble in the past has been that the veneer that covers Chinese personality has been accepted without seeking deeper for its primitive foundation.

It is hard to say in a few simple words what nervousness is, but it may be defined as a want of mental balance and self-control, an inability to muster one's mental and physical resources to meet the demands of circumstances, a tendency to neuropsychosis. Measured by this standard, the Chinese are a nervous people. An example is the want of self-control in their cyclonic outbursts of passion, the abandon of their grief, the way in which crowds are easily swayed and the hysterical condition of the whole people in times of national disaster. As to nervous and mental diseases in China, they are not uncommon; the political and social changes through which China is now passing, the impact of civilization, the incessant influx of new and disturbing ideas and the keener and wider competition in business will make these diseases still more common in the future.

Correspondence

SOME ERRORS IN DR. FRANK FREMONT-SMITH'S ARTICLE "THE NATURE OF THE CEREBROSPINAL FLUID"

To the Editor:—In referring to my views on the source of the cerebrospinal fluid, Dr. Frank Fremont-Smith (ARCH. NEUROL. & PSYCHIAT. **17**:317 [March] 1927) made some erroneous and misleading statements which I feel must be corrected. He states: "The following is an example of the type of evidence, derived from pathologic studies that he (Hassin) offers to support his contention (that the choroid plexus does not produce the cerebrospinal fluid): A case of carcinoma of the breast showed diffuse carcinomatosis of the meninges and a mass of carcinoma cells in the choroid plexus." This "type of evidence" was not offered by me. The choroid plexus changes to which Dr. Fremont-Smith refers were described, jointly with Singer, in a case of cerebral carcinoma (Histopathology of Cerebral Carcinoma, ARCH. NEUROL. & PSYCHIAT. **8**:155 [Aug.] 1922), while the "diffuse carcinomatosis of the meninges" was from a different case described in 1919 (Carcinoma of the Cerebral Meninges, ARCH. NEUROL. & PSYCHIAT. **1**:705 [June] 1919). Both these articles were referred to in a paper "Notes on the Nature and Origin of the Cerebro-Spinal Fluid" in the *Journal of Nervous & Mental Disease* (**59**:113, 1924), one of the three articles Dr. Fremont-Smith used as references.

He also makes the equally unfounded statement that "unfortunately he gives no evidence to prove this point so vital to his theory" (invasion of the cerebral meninges by carcinoma cells, by direct extension along the lymphatics of the neck). Such evidence is given by me in detail in the article on meningeal carcinoma.

Having confused the two cases, Dr. Fremont-Smith proceeds to make still other incorrect statements, as follows: "He believes that the presence of tumor cells in the choroid plexus indicates that the cerebrospinal fluid flows from the subarachnoid space into the ventricles and is absorbed by the choroid plexus." I cannot conceive why I should be held responsible for such nonsense.

Dr. Fremont-Smith gives an explanation of his own for the possible invasion of the choroid plexus by carcinoma. Here he has in mind my case of meningeal carcinoma secondary to a primary carcinoma of the breast; in my report of this case, the condition of the choroid plexus is not mentioned at all. But he assumes that a "blood borne metastasis, either passing through the lung or arising from a metastasis in the lung (no report of the lungs is furnished in Hassin's paper) reached the choroid plexus, from which tumor cells escaped into the ventricle and thence were distributed throughout the subarachnoid space." The condition of the cavity of the chest was given in my paper, "Carcinoma of the Cerebral Meninges" in 1919, while the mode of invasion of the choroid plexus occurred in an entirely different case and was given a proper interpretation by Singer and me in the paper on cerebral carcinoma in 1924.

Dr. Fremont-Smith summarily dismisses other facts furnished by pathologic studies, such, for instance, as "pigment" and "lipoid" accumulation in the cells of the choroid plexus. He merely states that the histopathologic evidence

offered by me is altogether uncertain, is "open to more than one interpretation" and therefore "cannot stand against the crucial experiments of Weed or Dandy."

To this I must say that the experimental evidence is much more uncertain than the pathologic; it is also open to more than one interpretation; the latter (pathology), if properly studied and understood, can stand against any experiments, however skilfully performed. Dr. Weed's remarkable work has always been referred to in my contributions, including those Dr. Fremont-Smith used as references, while I discussed Dandy's "crucial" experiment at the fifth annual meeting of the Association for Research in Nervous and Mental Diseases, in 1924, at which Dr. Fremont-Smith was present. In the volume on cerebrospinal fluid issued by that association, Dr. Fremont-Smith will find in the discussions that even crucial experiments like those of Dandy are open to various interpretations. My contribution presented at that meeting "Effect of Organic Brain and Spinal Cord Changes on Subarachnoid Space, Choroid Plexus and Cerebrospinal Fluid" has also been published in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY (**14**:468 [Oct.] 1925) but without the discussions. That paper, merely a short review of a number of pathologic facts recorded by me in various publications, was the main source from which Dr. Fremont-Smith drew his conclusions concerning the histopathologic evidence that the choroid plexus is an organ of excretion, not of secretion. If he had read the few references he gives more carefully he probably would not have ascribed to me statements I never made.

G. B. HASSIN, M.D., Chicago.

DR. FRANK FREMONT-SMITH'S REPLY TO DR. HASSIN'S CRITICISM

To the Editor:—Dr. J. B. Hassin's Criticism of my paper "The Nature of the Cerebrospinal Fluid" (ARCH. NEUROL. & PSYCHIAT. **17**:317 [March] 1927) has been referred to me for reply. It appears that I have confused a case of cerebral carcinoma with one of meningeal carcinoma, thereby misrepresenting Dr. Hassin in regard to these two cases. I apologize to Dr. Hassin for this mistake.

The point I wished to make is that the presence of tumor cells in both the brain substance and the choroid plexus is not good evidence that the tumor cells were carried to the choroid plexus by the flow of cerebrospinal fluid, for such cells may have reached the choroid plexus by way of the blood stream. In describing a case of sarcoma of the brain in the region of the third ventricle, in which the choroid plexuses of the lateral ventricles were infiltrated with sarcoma cells, Dr. Hassin says: "The flow of pathologic cell bodies from the parenchyma of the brain through the perivascular spaces to the ventricles and choroid plexus is thus most clearly depicted." This type of evidence does not support Dr. Hassin's contention that the choroid plexus "has absolutely nothing to do with the elaboration of the spinal fluid." These quotations are taken from Dr. Hassin's paper and discussion (Chapter XXIII in "The Human Cerebrospinal Fluid," Association for Research in Nervous and Mental Diseases, New York, Paul B. Hoeber, 1926, vol. 4).

In this connection, I would recall an observation made by Cushing, who saw a clear fluid coming from the surface of a choroid plexus, exposed in the course of an operation. When the blood supply to the choroid plexus was ligated, the formation of fluid immediately ceased. Schaltenbrand and I. Putnam, in recent experiments at the Harvard Medical School, have studied through the microscope the choroid plexus of living cats. Immediately following the intravenous injection of fluorescin, they observed fluid colored with the

dye issuing abundantly from the choroid plexus while only small amounts of fluorescin escaped from the vessels of the meninges (*Deutsche Ztschr. f. Nervenh.* **96**:123, 1927).

These direct observations, together with the experiments of Weed and Dandy, referred to in my paper, justify the conclusion that the choroid plexus is the chief source of the cerebrospinal fluid.

F. Fremont-Smith, M.D., Boston.

News and Comment

THE INTERNATIONAL LEAGUE AGAINST EPILEPSY

A meeting of the Ligue Internationale Contre L'Epilepsie (The International League against Epilepsy) will be held in Zurich, Switzerland, early in August, 1927. The exact date will be announced later. Those who wish to present communications or to read papers are invited to communicate with Dr. L. J. Muskens, secretary, 136 Volden Straase, Amsterdam, Holland; Dr. Ulrich, Canton Interspital, Zurich, Switzerland; Dr. G. Kirby Collier, 80 East Avenue, Rochester, N. Y., or Dr. A. L. Shaw, The Olbiston, Utica, N. Y.

Abstracts from Current Literature

THE ALLOCORTEX IN ANIMALS AND IN MAN. MAXIMILIAN ROSE, J. f. Psychol. u. Neurol. **34**:1, 1926.

The allocortex is phylogenetically an old region. A study of its development in mammals as well as in the lower vertebrates seemed, therefore, a fruitful field for investigation, and this contribution represents the author's masterly research in this direction. The paper (111 pages, with 21 illustrations in the text and 30 double plates) contains the results of Rose's investigation of the allocortex in the various orders of birds and reptiles, in monotremata, marsupialia, chiroptera, insectivora, rodentia, carnivora and prosimii. Special attention is given to the areas generally regarded as functionally cooperative, such as the bulbus olfactorius, regio prepyriformis, regio periamygdalaris, regio entorhinalis and cornu ammonis — all of which are supposed to subserve the sense of smell.

On embryologic and comparative anatomic grounds, Rose concludes that the laws of development of the mesocortex and allocortex are the same in mammals as in man, although they are different as far as their individual cortical areas are concerned.

It is generally accepted that certain parts of the secondary cerebral vesicle give rise to the striatum, while others give rise to the cerebral cortex. In mammals it is principally the basal, and in birds and reptiles the basal and lateral walls of the secondary cerebral vesicle that give rise to the striatum. Sometimes it is extremely difficult to distinguish definitely between cortex and striatum. This is especially so in the lower vertebrates.

Ontogenetic studies would seem to indicate that the cortex and striatum are closely related to each other, because both are differentiation products of the wall of the secondary cerebral vesicle.

The author next points out the extreme circumstances under which one part of the wall of the secondary cerebral vesicle develops exclusively into a cortex, while another part develops exclusively into a striatum. In the first case, there is formed the so-called cortical plate (protoptyx). During a certain ontogenetic period, these parts of the wall of the secondary cerebral vesicle develop into a matrix, an intermediary layer, a cortical plate and a marginal layer. The further development of this cortex depends in principle on a more or less progressive stratification of the cortical plate. The marginal layer in its fully developed state becomes the zonal layer—a layer that is either poor in cells or completely acellular; it is found in every type of cortex. The parts of the secondary cerebral vesicle which form the striatum exclusively never give rise to a true cortical plate with its stratification, nor to a zonal layer. With these facts in mind, Rose's definition of the cortex is as follows: "The cerebral cortex is that product of the wall of the secondary cerebral vesicle which shows the presence of pyramidal cells and a horizontal arrangement of layers, in which the zonal layer is never missing." The striatum, on the other hand, is "that product of the wall of the secondary vesicle which shows no such characteristics of a cortex." This definition does not include every characteristic of the cortex, and therefore must not be regarded as complete. It merely indicates the morphologic criteria by which the cortex can be distinguished, and in this way serves as a sound working basis for the purposes of this discussion.

A cortex so defined does not show a uniform mode of development. The most primitive mode of cortical development is represented by the semiparietine or striatal cortex. In the development of this type of cortex the entire cellular layer of the matrix, which actually forms the striatum, does not participate, and therefore the cortex in these parts does not represent the product of the entire wall of the secondary cerebral vesicle—it is only a part of it. It is for this reason that this type of cortex is designated as a semiparietine cortex, in contradistinction to totoparietine cortex in the formation of which the entire cellular layer of the matrix is found to participate. The semiparietine cortex is also designated as the striatal cortex, because it is situated on the surface of the striatum and is closely related to it ontogenetically. According to Rose, the semiparietine cortex includes the regio prepyriformis, the regio periamygdalaris, the tuberculum olfactorium, the septum pellucidum and the area diagonalis. It contains, therefore, a number of cortical areas, all of which go through the same mode of primitive development, based on the principle that the striatum as well as the cortex is developed from the same matrix in the same part of the secondary cerebral vesicle.

The cortex totoparietinus consists of two groups of cortexes. In the first group there arises, during a certain period of its development in the region of the wall of the secondary cerebral vesicle, a matrix, an intermediate layer, a cortex plate (protoptyx) and a marginal layer. This is followed by a successive migration of the cells of the matrix through the intermediary layer into the protoptyx; stratification, however, does not occur until the latter (protoptyx) is reached. All cellular layers of this type of cortex are derived from the protoptyx; the latter is, therefore, designated as "cortex holoprotoptychos."

In the second group of totoparietine cortexes occurs a different type of layer formation. Here the wall of the secondary cerebral vesicle also gives rise, in the beginning, to a matrix, an intermediary layer, a cortical plate and a marginal layer, but the cortical plate itself receives only a limited number of cells from the matrix. Owing to this, the protoptyx is small. The elements which later migrate from the matrix do not reach the cortical plate itself but form an accessory cortical plate underneath the cortical plate proper. This accessory cortical plate is separated from the true cortical plate by a pale, almost cell-free, zone, which in the course of further development remains either undivided, just as the cortical plate proper, or else it goes on to the formation of a more or less advanced stratification. In contradistinction to the holoprotoptychos type of cortex, this type of cortex is designated as a "schizoprotoptychos cortex," i. e., one derived from a double (divided) cortical plate, whereas in the holoprotoptychos type of cortex all cellular layers are derived from the cortical plate.

A primitive schizoprotoptychos cortex is made up of the following layers: (*a*) lamina zonalis; (*b*) lamina principalis externa; (*c*) lamina dissecans and (*d*) lamina principalis interna. The lamina principalis externa corresponds to the external (genuine) cortical plate, and the lamina principalis interna to the internal (accessory) layer. The lamina dissecans represents the acellular, or almost acellular layer which separates the cellular layers mentioned in the foregoing from one another.

This four-layer type of cortex is the tectogenetic basic type of the schizoprotoptych cortex. In some types of cortex (for example, in the area presubicularis), the tectogenetic basic type of a schizoprotoptych cortex is the permanent type of cortex; in other types, however, the lamina principalis externa or interna

goes on to more or less stratification, which occasionally gives rise to a marked increase in the number of layers.

It is of interest to note that the schizoprotoptych cortex follows the same laws of development in all species from the lowest mammal to man; as a matter of fact, the same mode of development of this cortex can be demonstrated in the lower vertebrates (some birds and reptiles). The same is also true of the development of the semiparietine cortex. From a developmental point of view the cortex semiparietinus may be regarded as the most primitive cortex and the schizoprotoptych cortex as one of much higher development.

In studying the histogenetic principles involved in the various parts of the cerebral cortex in mammals and in man, Rose finds that during a certain ontogenetic stage the wall of the secondary cerebral vesicle shows stratification in those parts, which later go on to the formation of the cornu ammonis, subiculum, fascia dentata, tenia tecta (i. e., the entire ammonis system), the area retrobulbaris, the area limbica anterior, the area retrosplenialis granularis and agranularis, and the entire isocortex. He differentiates the following principal layers: cortical layer, cortical plate (protoptyx), intermediary layer and the matrix; in man, the intermediary layer consists of several transitional layers.

In the course of further ontogenetic development the matrix constantly diminishes in size, its cells contributing to the structure of the cortical plate, until finally there is a complete disappearance of the matrix and intermediary layer. Up to a certain stage of development, the cortical plate remains undivided in the region of the entire holoprotoptych cortex. During this stage, there is only a quantitative difference between the so-called homogenetic cortex and the formatio ammonis, the difference being in the general narrowness of all layers of the wall of the hemisphere and especially of the cortical plate in the region of the formatio ammonis. This is the case in animals as well as in man. During the period of still further ontogenetic development, it is found for the first time that the homogenetic cortex, the area limbica anterior, the area retrosplenialis granularis and area retrosplenialis agranularis become separated from the formatio ammonis, so that, in the lower animals as well as in man, the cortical plate in the region of the homogenetic cortex, the area limbica anterior, retrosplenialis granularis and agranularis, is divided into four layers, whereas the cortical plate in the formatio ammonis remains undivided. When the lamina zonalis is added to these four layers, there are five layers in the parts of the cortex described: (*a*) lamina zonalis (I); (*b*) lamina granularis primaria (later II-IV); (*c*) lamina ganglionaris (V); (*d*) lamina multiformis (VI); (*e*) lamina infima (VII). At this ontogenetic stage, then, the I, V, VI and VII layers of Brodmann's tectogenetic basic type of cortex are already well established in these areas, whereas Brodmann's II, III and IV layers are represented only by an undifferentiated layer consisting of granular elements (lamina granularis primaria).

During this same period of development, the formatio ammonis consists of only two layers—(*a*) lamina zonalis (I), and (*b*) cortical plate—but the differentiation of the pyramidal cells occurs early.

During further ontogenetic development (from six to seven months in the human embryo) the holoprotoptych cortex undergoes a second developmental change. At this time the original granular layer in a large portion of the homogenetic cortex becomes split up into three parts. This division gives rise to the well known seven-layer tectogenetic basic type of cortex. The cortex of the area limbica anterior, area retrosplenialis granularis and agranularis,

however, does not keep pace with this further differentiation of the original granular layer; owing to the nondivision of the lamina granularis primaria, it remains a five-layer cortex, but the formatio ammonis remains a two-layer cortex, even in this ontogenetic stage.

A study of the further development of these parts of the cortex reveals that the aforementioned principle of division into layers persists throughout the remaining periods of development. The formatio ammonis always remains a two-layer cortex in which the cells of the cortical plate only go on to differentiation into pyramidal cells. Its cellular layer corresponds to all the cellular layers of the isocortex, because the cortical plate in the region of the isocortex has become subdivided into six layers. This fact was established by Rose first in mammals and later in man. Previous observers were inclined to regard the cellular layer of the cornu ammonis as the homolog of the II, V or the VI layer of Brodmann's tectogenetic basic type of cortex.

The cortex of the area limbica anterior and area retrosplenialis granularis and agranularis also shows a constant mode of development — principally a five-layer development. The lamina granularis primaria in the area retrosplenialis granularis remains in many animals and in man granular throughout life, but in some species (rabbits) a layer of elements lying closely under the zonal layer differentiates into smaller or larger pyramidal cells. The area limbica anterior and the area retrosplenialis agranularis are characterized by the fact that all the cells of their original granular layer are differentiated into small and moderately large pyramidal cells which form a dense layer under the zonal layer constituting a sort of secondary layer.

Thus it can be seen that the holoprotoptych cortex in its final development includes a number of entirely different types of cortexes which, however, have one important genetic characteristic in common, i. e., that all cellular layers of these types arise from the cortical plate. In addition to the individual differentiation of their cells, the degree of differentiation of these cortexes finds special expression in the stratification of the cortical plate. The formatio ammonis undoubtedly represents a most primitive type of development in which stratification of the cortical plate has never occurred; it represents the cortex holoprotoptychos bistratificatus. The highest stage of cortical differentiation is represented by the homogenetic cortex (isocortex), in which the cortical plate is stratified into six layers. The area limbica anterior and the area retrosplenialis granularis and agranularis with a division of the cortical plate into four layers occupy an intermediary position between these two types of cortical development.

Genetically then, the cortex may be divided as follows:

I. Cortex semiparietinus (striatalis). Regio prepyriformis, tuberculum olfactorium, regio periamygdalaris, septum pellucidum, area diagonalis.

II. Cortex totoparietinus.

A. Schizoprotoptychos

(*a*) Parumstratificatus (area presubicularis, area parasubicularis, area perirhinalis),

(*b*) multistratificatus (regio entorhinalis),

B. Holoprotoptychos

(*a*) bistratificatus (cornu Ammonis, subiculum, tenia tecta, fascia dentata, area retrobulbaris),

(*b*) quinquestratificatus (area limbica anterior, area retrosplenialis granularis, area retrosplenialis agranularis),

(*c*) septemstratificatus (isocortex euradiatus).

Only the cortex holoprotoptychos septemstratificatus corresponds to O. Vogts' isocortex euradiatus or to Brodmann's homogenetic cortex. The cortex holoprotoptychos quinquestratificatus, having never gone through a six or seven-layer formation, cannot be said to be included in the isocortex euradiatus; owing to its relatively far advanced differentiation into layers, it cannot be included in the allocortex, and being an intermediate stage between the cortex holoprotoptychos bistratificatus and the cortex euradiatus, it is designated as the mesocortex. The isocortex and mesocortex are characterized by the fact that, genetically, they both represent one mode of development, whereas the allocortex represents several modes of development.

After describing in great detail the comparative anatomy of the areas enumerated in the foregoing in marsupialia, monotremata, insectivora, chiroptera, rodentia carnivora and prosimii, the author makes some interesting observations as to the functions of these areas from a morphologic point of view.

CORTEX SEMIPARIETINUS (STRIATALIS)

The *regio prepyriformis* (palaeocortex, A. Kappers) is phylogenetically the oldest cortex; its mode of development is primitive, being derived from the cells of the same matrix as the striatum. It is not found in all birds; its homolog could not be found in singing birds (*Oscines*), nor could it be found in the parrot (*Psittacidae*). It is unusually well developed in swimming birds (*Natatores*). In ducks (*Anas domestica*), it is situated on the mesial surface of the frontal pole and close to the striatum; in the polar diver (*Colymbus arcticus*), this region is as well developed as in ducks but is located on the lateral surface of the frontal pole. It is less extensive in pigeons (*Gyrantes*) than in swimming birds, although it is prominent, with its greatest part on the basal surface of the frontal pole. It is somewhat less extensively developed in climbing birds (*Scansores*). It is prominent in hens (*Rasores*), birds of prey (*Rapaces*) and waders (*Grallatores*), and is also situated on the basal surface of the frontal pole. The same is true of *Levirostres* and of *Upupa epops*. There is barely an indication of it in swifts (*Cypselus apus*). In the birds examined, the regio prepyriformis showed no differentiation into subareas. Rose believes that birds without a regio prepyriformis do not possess an olfactory function — a function usually attributed to this area. The quantitative components of this function are in direct proportion to the extent of development of the area prepyriformis.

In contrast to birds, all reptiles show a comparatively well developed area prepyriformis; in a vast majority of them it occupies the entire surface of the frontal hemispheres but diminishes in size caudad. There is no differentiation into subareas, and the type of development is the same in all reptiles. Generally speaking, the extent of this area in these animals is also in direct proportion to the size of the olfactory bulb.

The regio prepyriformis shows great variations both as to extent and location in the different orders of mammals. In the duck-mole (*Ornithorhynchus*), phylogenetically the lowest mammal, this region, and for that matter the entire allocortex, occupies a relatively small part of the forebrain; in this animal, the bulbus olfactorius is also relatively small. The claim that phylogenetically lower mammals have a relatively larger archipallium (allocortex) than phylogenetically higher mammals apparently does not apply to the *Ornithorhynchus*. Notwithstanding the small size of the area in this animal, it is differentiated into three unusually small subareas: area prepyriformis lateralis, intermedia and medialis.

In the marsupialia (*Didelphys*) this region is also subdivided into three subareas, which are extensive in size and in proportion to the extensive development of the bulbus olfactorius; the latter is more than twice the size of the regio prepyriformis.

In the chiroptera (only *Vesperugo pipistrellus* and *Vespertilio murinus* were examined), the regio prepyriformis is also divided into three subareas, the whole region being small.

In the rodentia (mouse, guinea-pig, coney) this region is divided into four comparatively well developed subareas: area prepyriformis lateralis, intermedia, medialis and ventralis.

The regio prepyriformis is also subdivided into four areas in the insectivora (*Erinaceus europeus, Talpa europea, Sorex vulgaris*). Its size and surface development go hand in hand with those of the bulbus olfactorius; in the hedgehog, the bulbus olfactorius is almost twice the size of the regio prepyriformis.

In carnivora (the dog) the regio prepyriformis is divided into five subareas: area prepyriformis lateralis, intermedia, medialis, basalis alpha, and basalis beta. Although extensive in size, it is much smaller than the bulbus olfactorius, which is about one and a half times as large as the former.

Considerable difficulties are encountered in studying this region in monkey and in man; the brain of the lemur still offers opportunities for comparative study. In this animal, which stands midway between the lower mammals on the one hand and monkey and man on the other, the entire regio prepyriformis, in contrast with that of the dog and the other lower mammals, occupies the basal surface of the forebrain and is subdivided into four areas only. The area prepyriformis is especially complicated in the primates. In the chimpanzee, it is situated on the basal surface of the frontal lobe forming a transition between this surface of the frontal lobe and the area periamygdalaris in the temporal lobe. The area prepyriformis medialis in primates corresponds to the so-called gyrus olfactorius lateralis. In man, the anatomic conditions are precisely the same as in the monkey. It must here be reiterated that in lower mammals the size of the surface of the bulbus olfactorius is considerably larger than that of the regio prepyriformis; this is so in the half-apes, though not to the same extent as in the marsupialia and insectivora. In animals without a bulbus olfactorius (singing birds and parrots), no regio prepyriformis can be found.

Tuberculum Olfactorium: Birds do not show any homolog of the tuberculum olfactorium. There is no uniformity in its formation in reptiles; whereas it is prominent in crocodiles and in *Chelonia* (turtles), its presence cannot be definitely established in *Lacertilia* (lizards) and *Ophidia* (snakes). There is no differentiation into subareas in crocodiles and turtles. It is prominent in all mammals and in man; it is well developed in all lower mammals and is differentiated into two or three subareas, but is poorly developed in microchiroptera and in beaked animals. It is as prominent in the half-apes as in the lower mammals, but its architectonic characteristics are not as well marked as in man and in apes. In the former, the anterior portion of the substantia perforata anterior (eminentia parolfactoria, Beccari) corresponds to the tuberculum olfactorium or lobus parolfactorius. In man, its absolute size is considerable and it is subdivided into three subareas. All animals possessing a well developed tuberculum olfactorium also have a bulbus olfactorius, although some animals with a well developed bulbus olfactorius have no tuberculum olfactorium (birds).

Area Diagonalis: Owing to the large size of its cells, the area diagonalis could be demonstrated in all mammals. Its position behind the tuberculum olfactorium is constant, and it always ascends on the mesial surface of the brain in the form of a small band to the level of the corpus callosum (tenia tecta). The tenia tecta is in all mammals on the posterior portion of the corpus callosum. At the genu it bends ventrad until it reaches the rostrum, where it is found below and anteriorly, terminating in the area retrobulbaris. In lower mammals, its presence can only be determined architectonically, because after it leaves the rostrum it ceases to be clearly visible macroscopically.

In apes, the descending part of the tenia tecta appears as a definite gyrus corresponding to the so-called gyrus olfactorius medialis. In man this convolution is less distinct, and terminates, as in lower mammals, in the area retrobulbaris. In lower mammals the area diagonalis lies behind the tuberculum olfactorium and extends in the form of a band to the mesial surface of the hemisphere. In monkeys and in man, there is also a distinct convolution behind the tuberculum olfactorium, which also extends to the mesial surface of the cerebral hemisphere. This convolution (gyrus circumflexus, or the gyrus subcallosus of Economo and Koskinas) bears no relation to the tenia tecta or to the induseum.

Septum Pellucidum: This structure is found in all birds and reptiles; it is a well defined type of cortex in all mammals. Economo and Koskinas do not include the septum pellucidum in the human cortex.

Regio Periamygdalaris: In its final development the regio periamygdalaris is closely connected with the nucleus amygdalae; it is a striatal cortex derived from the same matrix as the nucleus amygdalae. It has no homolog in birds and reptiles; in these animals, the epistriatum (Edinger, Kappers) corresponds to the nucleus amygdalae in mammals. The regio periamygdalaris is divided in the monotremata (*Ornithorhynchus*) into two subareas; it is much more extensive in marsupialia than in beaked animals, and is subdivided into three areas, as it is in chiroptera and insectivora, except in the mouse in which it has only two subareas. This region is especially well marked in the dog in which it also consists of three subareas. In all the animals mentioned, this area is on the basal surface of the forebrain posterior to the regio prepyriformis; caudad to the regio periamygdalaris is the regio entorhinalis. In all mammals the regio periamygdalaris extends from the mesial surface of the forebrain to the formatio ammonis. In the higher mammals (ascending from the lemur) the regio periamygdalaris, owing to the marked development of the temporal lobe, is displaced together with the regio entorhinalis to a frontodorsomesial position, so that in the lemur it is found on the mesial surface of the temporal pole. In half-apes this region is divided into four subareas. Hand in hand with the increased development of the temporal lobe in a frontal direction, the regio periamygdalaris undergoes a displacement from the basal surface of the forebrain to the dorsomesial surface of the temporal lobe. In the chimpanzee, this region is divided into five subareas. In man the area periamygdalaris is found exclusively on the dorsomesial surface of the temporal lobe; it is an isocortex and is divided into four subareas. The gyrus semilunaris (Retzius) is especially prominent in man. This area is included by Economo and Koskinas in their area pyriformis temporalis. In apes and in man as well as in the lower mammals, the regio periamygdalaris extends anteriorly into the regio prepyriformis and caudad and mesially into the cornu ammonis. In the lower mammals, the regio periamygdalaris is almost always less extensive than the bulbus olfactorius. In primates this relationship is just the opposite.

Bulbus Olfactorius: In vertebrates, the bulbus olfactorius shows great developmental variations. In the lower mammals there is some parallelism between its development and that of the regio prepyriformis. In parrots it is two-paired but is so small that it is difficult to find its homolog. It is also rudimentary in singing birds, granivora and insectivora; in these animals it is unpaired, small and completely covered by the frontal pole, as in parrots. It is better developed in hens, pigeons, birds of prey, and climbing birds; in these, it is relatively large, paired but still covered by the frontal pole. It is well developed in swimming birds, and is almost of the same size in the polar diver as in the rodents; it is paired and protrudes beyond the frontal pole. It is also paired in the *Upupa epops* but is rudimentary and is unpaired in the *Cypselus apus* (these belong to the order of *Levirostres*). A well developed bulbus olfactorius is found in all reptiles. It is paired and well developed in all mammals; in these are found variations only in size but not in structure. It is insignificant in the *Ornithorhynchus*, and is considerable in size in the marsupialia; it is smaller in bats than in the opossum; rodents (mouse, guinea-pig) have a smaller bulbus olfactorius than marsupialia, but a larger one than bats; it is large in insectivora but smaller than in the didelphys. It is large in the dog and relatively large in the half-apes, though smaller than in the insectivora and marsupialia. Its structure is different in the apes than in other mammals; in the former, the olfactory fibers are found in the inferior surface of the bulbus, whereas in the other lower mammals the fila olfactoria occupy the entire bulb, so that in apes only the inferior surface must be regarded as the genuine bulbus olfactorius. It is somewhat larger in man than in the chimpanzee and the fila olfactoria are only found on the inferior surface of the bulb. It is small in primates and in man; in the hedgehog it is one third and in *Didelphys* double the size of that in man; it is more than six times as large in the dog as in man. In lower mammals, the bulbus is always larger than the regio prepyriformis; it is almost double the size of the latter in the common hedgehog and in the dog and more than double in *Didelphys*. In half-apes the size of the bulb is almost 50 per cent larger than in the area prepyriformis.

CORTEX HOLOPROTOPTYCHOS BISTRATIFICATUS

This cortex consists of the subiculum, the cornu ammonis with its five subareas, the tenia tecta, the fascia dentata and the area retrobulbaris; all of these with the exception of the area retrobulbaris constitute the formatio ammonis.

In birds the formatio ammonis is best developed in *Rapaces, Gyrantes* and *Natatores*, but it shows no differentiation into subareas. It occupies in the caudal portion of the forebrain the entire mesial, and part of the ventral, portion of the wall of the ventricle, and ventrad it gradually becomes separated from the basal wall of the latter. It is not rolled up, as in mammals, but is freely exposed on its surface. It is well marked in *Grallatores, Rasores* and *Scansores* but occupies a smaller surface than in *Rapaces, Gyrantes* and *Natatores*. There is no absolute parallelism between the size of the bulbus olfactorius and the area prepyriformis on the one hand, and the formatio ammonis on the other. In singing birds, the formatio ammonis is so rudimentary that it can hardly be recognized. No homolog of this structure can be found in parrots. Reptiles show a well developed formatio ammonis, but it shows great variations in size and position; in some of these it is subdivided into a number of subareas. Although subareas III and IV are double-layered in

reptiles, nevertheless they probably represent in mammals the region that gives rise to the isocortex, whereas area V (of the formatio ammonis) is the prototype of the entorhinal cortex. It is also noteworthy that areas II, III and IV of the formatio ammonis are situated between the cornu ammonis (area I) and the regio prepyriformis (Brodmann's area 51), so that they occupy the same position as the isocortex in mammals.

In *Ornithorhynchus*, an animal without a corpus callosum, the formatio ammonis occupies the mesial surface of the hemisphere, extending ventrad to the area retrobulbaris and dorsad to the area presubicularis or entorhinalis; only its most anterior portion occupies the dorsal part of the area limbica anterior, whereas its caudal portion merges into the area periamygdalaris. Conditions here are relatively simple, because owing to the absence of a corpus callosum there is no tenia tecta. The subiculum, cornu ammonis and fascia dentata extend in the form of a wide arch from the area periamygdalaris almost up to the frontal pole; at the same time, the fascia dentata appears throughout its entire extent free on the mesial surface of the cerebral hemisphere, whereas the sulcus hippocampi causes the cornu ammonis and subiculum to be rolled up into a fold; only in the most anterior part of the brain can the cornu ammonis be seen without the fascia dentata lying freely on the surface and above the septum pellucidum. The part of the cornu ammonis which extends directly into the area retrobulbaris reminds one of the tenia tecta in other mammals. In *Ornithorhynchus*, the subiculum is remarkably small. The cornu ammonis is subdivided into three subareas, with the second of these forming the bulk of this structure. A large subarea II with a smaller subarea I and subiculum is characteristic in smaller mammals. In *Didelphys* (also devoid of a corpus callosum) the formatio ammonis is similar to that of *Ornithorhynchus;* the subiculum also is small. In these animals, the cornu ammonis is divided into five subareas, the first of which is smaller than in *Ornithorhynchus*, whereas the second is several times larger. Owing to the character of the cells, the third and fourth subareas are sharply outlined; the fifth area represents the fascia dentata and is as well developed as in beaked animals.

In microchiroptera (*Vesperugo pipistrellus*), the cornu ammonis together with the fascia dentata begins in the regio periamygdalaris, whence it turns downward in the form of an arch to reach the dorsal surface of the corpus callosum. It is subdivided into three subareas. Area I is the most extensive and is characterized by the fact that the pyramidal cells are densely packed together under the zonal layer but become scattered far apart as one follows the area ventrad; area II is relatively smaller, and its cells are few in number and not quite as epithelial as in *Didelphys*; the subarea found in the fascia dentata corresponds to area V in the other mammals. Compared to *Didelphys*, the subiculum is larger, relatively, as well as absolutely.

In insectivora (animals with a well formed corpus callosum), the cornu ammonis is situated only in the caudal part of the hemisphere ventrad to the corpus callosum, the subiculum, cornu ammonis and fascia dentata being rolled up in the ventricle. At the splenium, the cornu ammonis extends directly into the tenia tecta which runs along the dorsal surface of the corpus callosum to the frontal pole, covering the genu until it reaches the rostrum, where it bends first ventrad, and then forward to merge with the area retrobulbaris.

In the hedgehog (and in all small mammals), the subiculum accompanying the cornu ammonis and the tenia tecta throughout its entire extent is relatively

small; it is divided into five subareas, the first of which is relatively small, being markedly exceeded in size by the second subarea, whereas the third, fourth and fifth are unusually well developed. In rodents (mouse), the cornu ammonis without the subiculum consists of four subareas, the first of which, though relatively small, is well marked; areas II and III are also distinct.

In all mammals the lower part of the cornu ammonis adjoining the regio periamygdalaris is a primitive structure; it is not differentiated into any subareas.

In the dog, the cornu ammonis is divided into five subareas, which resemble in size and architectonic structure those of man. The subiculum is also unusually well developed. In the hedgehog, there is a direct merging of the tenia tecta into the cornu ammonis. In the dog, the subiculum runs closely underneath the splenium and extends anteriorly where is forms the gyrus subicularis; it is differentiated into two subareas, a ventral and a dorsal.

In half-apes, apes, and man the formatio ammonis also consists of five subareas.

The zonal layer in the cornu ammonis represents ontogenetically the marginal layer just as in the isocortex; the lamina zonalis in the former is the exact homolog of the lamina zonalis in the latter. Embryologically, the cellular layer of the cornu ammonis represents an unstratified cortical plate. In the isocortex, the same plate develops into six layers; it is therefore impossible to homologize (as Economo and Koskinas have recently attempted) the cellular layer of the cornu ammonis with any of the layers of the isocortex, because on ontogenetic as well as comparative anatomic grounds, this cellular layer really corresponds to all layers of the isocortex.

Generally speaking, the subiculum and its first subarea is poorly developed in the lowest mammals and is well developed in the higher; these areas are of considerable size in the dog and are well developed in apes and in man. Animals with small subicula and subareas I usually have much larger subareas II, III and IV, while the reverse of this is observed in animals with larger subicula and subareas I.

The size of the bulbus olfactorius in lower mammals is greater than the size of the cornu ammonis including the tenia tecta. Ascending the animal scale, there is a relative increase in the size of the cornu ammonis. When the size of the entire semiparietine cortex including that of the bulbus olfactorius is compared with that of the cornu ammonis, the former is found to exceed the latter in lower animals, whereas in apes and especially in man, conditions are just the opposite. There is no doubt that the bulbus olfactorius is the first olfactory station; the semiparietine cortex is generally regarded as the second station (palaeopallium, A. Kappers); many authorities consider the cornu ammonis a third station (archipallium, A. Kappers). If this is so, the morphologic facts described assume no little significance. Rose could establish no parallelism between the size of the bulbus olfactorius and the cortex semiparietinus on the one hand and the cornu ammonis on the other. The fact that in man and in apes the cornu ammonis is much larger than the bulbus olfactorius combined with the semiparietine cortex can be interpreted in no other way than that the cornu ammonis must receive other fibers than those assumed by previous investigators to be olfactory fibers of the third order. This naturally raises the question whether the cornu ammonis does not subserve in man and in apes other than olfactory functions. The answer to this question needs further investigation; here it is only necessary to point out what deductions one may be able to make from study based purely on morphology.

Fascia Dentata: No fascia dentata could be found in birds. Its development is not uniform in reptiles; it is well developed in *Lacertilia* and *Ophidia* in which it is found exclusively on the surface of the hemisphere in its mesiodorsal angle; in some reptiles (snakes) it is merged with the cornu ammonis; no homolog could be found in *Chelonia* and crocodiles. Conditions, on the other hand, are uniform in mammals, a well developed fascia dentata being found in all mammals from the monotremata to man. Whatever variations there may be in its location, size and relation to the cornu ammonis, area periamygdalaris and tenia tecta in the various mammals depend largely on the presence or absence of the corpus callosum in these animals. In all mammals, the cellular layer of the fascia dentata is to a great extent made up of granular cells. Embryologic investigation discloses that the cellular layer of the fascia dentata in mammals and in man consists, early in their ontogenesis, of large neuroblasts, which later undergo a secondary change during which they may become converted into granular cells. This leads Rose to conclude that, structurally, the fascia dentata represents a typical granular type of cortex.

Area Retrobulbaris: This type of cortex could be found in all vertebrates equipped with a well developed olfactory region, especially the regio prepyriformis. It is a two-layered cortex consisting only of a lamina zonalis and a moderately wide layer of pyramidal cells beneath the latter. As it constitutes the forward prolongation of the regio prepyriformis and of the tenia tecta in every type of cortex, it is regarded as an integral part of the formatio ammonis.

Only birds with a well developed regio prepyriformis and bulbus olfactorius have an area retrobulbaris. Its homolog could not be found in singing birds or parrots; it is small in *Levirostres* and barely demonstrable in *Longipennes* (gulls); it is well developed in *Natatores* and *Gyrantes*, but less so in *Cuculus canorus* (cuckoo) and *Accipiter nisus* (buzzards). Whereas in birds the entire area retrobulbaris lies in the region of the bulbus olfactorius, it is found in some reptiles also outside of the bulb; in the crocodile, for example, the area is so long that only its most anterior part is in the region of the bulb, while its greatest part runs freely for a considerable distance between the latter and the frontal pole. The same is true in *Lacertilia*. In *Ophidia*, it also runs for a considerable distance outside the bulbus. In *Chelonia* and in birds the entire area is within the bulb.

The area retrobulbaris is uniform in structure and position in the lowest mammals and up to the half-apes; it forms a prolongation of the prepyriform types and of the tenia tecta, and most of it lies in the region of the bulbus olfactorius. Identical conditions are found in apes and in man; in these the so-called tractus olfactorius corresponds genetically and architectonically to the area prepyriformis, its extensive area and elongated shape depending undoubtedly on the extensive development of the forebrain.

CORTEX TOTOPARIETINUS SCHIZOPROTOPTYCHOS

Area Presubicularis: This area is found in neither birds nor reptiles, but is present in all mammals, in some of which (half-apes, apes and man) it is subdivided into two subareas. It is in front of the subiculum in all mammals, and corresponds to the four layer tectogenetic basic type of a schizoprotoptych cortex.

Area Parasubicularis: This area represents the same type of a cortex as the area presubicularis, the only difference being that the lamina principalis externa in the area parasubicularis consists almost always of moderately large pyramidal cells, whereas in the area presubicularis it consists of a preponder-

ance of small nerve cells and granular elements. The area parasubicularis is found neither in birds nor in reptiles, nor is it always found in mammals. Although it occupies a definite gyrus in the dog, it is not conspicuous; it is small in the mouse. In most mammals, it is difficult to distinguish it from the entorhinal cortex.

Area Perirhinalis: This is a narrow, band-like area which forms a border around the lateral margin of the regio entorhinalis; it can be seen in all mammals, although it is more prominent in some than in others; it is especially prominent in the lower mammals, in which it can be seen freely on the surface because the sulcus rhinalis has not as yet completely dipped or has dipped only slightly into the deeper parts of the hemisphere. In the higher mammals, beginning with the dog, the area perirhinalis is found deeply within the sulcus rhinalis. This area almost always represents a typical four-layer type of schizoprotoptych cortex.

Regio Entorhinalis: This region is present in almost all birds; in them, its structure is characteristic and corresponds to a four-layer tectogenetic basic type of schizoprotoptych cortex. It is best developed in pigeons, singing birds, swimming birds and climbing birds; it is less marked in waders, hens, and birds of prey, and is still less developed in parrots. It is poorly developed in reptiles. Originally, this was a four-layered cortex; in addition to the zonal layer it contains two cellular layers separated from each other by a paler strip of cortex. This type of cortex is only found in *Varanus*—and in no other reptiles.

It represents a most constant type of cortex in all mammals, but varies in size in the different orders. In the lower mammals, its greatest part occupies the laterocaudal portion of the hemisphere, extending somewhat to the caudobasal and caudomesial surfaces; in *Ornithorhynchus*, however, most of the area is on the mesial surface of the hemisphere. With the gradually increasing development of the isocortex and especially of the temporal lobe, the entire entorhinal region is displaced, first basalward and later, mesially. The displacement toward the base is best seen in carnivora, whereas the displacement to the base and partly mesially is best seen in half-apes. In apes and in man, it is found almost exclusively on the mesial surface, and therefore bears a close relationship to the surrounding areas (regio prepyriformis, periamygdalaris, area perirhinalis, presubicularis and parasubicularis).

In birds and reptiles the region entorhinalis represents only one architectonic area—there is no differentiation into subareas; in mammals, however, there occur the greatest variations in the differentiation into subareas. In *Ornithorhynchus, Didelphys, Mus musculus, Erinaceus europeus* and *Vesperugo*, there are three subareas; the dog has no less than eight distinct subareas, half-apes eleven, apes, such as the chimpanzee, fourteen, and man twenty-three.

The entorhinal (hippocampal) cortex is generally regarded as an olfactory cortex; its function in the various orders of animals depends to a great extent on the number of architectonic subareas and the differentiation of the individual elements. The gyrus hippocampus in man, with his twenty-three subareas therefore represents a specific human organ.

Comparing the relative size of the entorhinal region with other regions of the allocortex, one finds the following: In lower animals (marsupialia, insectivora), the bulbus olfactorius is greater than the regio entorhinalis; in half-apes, the regio entorhinalis is greater than the bulbus; in apes and in man the entorhinal region is several times the size of the bulbus. Comparing the size of the entire cortex semiparietinus with that of the bulbus olfactorius

and entorhinal region, one finds that in the lowest animals the entorhinal region always exceeds the cortex semiparietinus and bulbus olfactorius — in some animals (*Didelphys*) several times; there is also a relative increase of the entorhinal cortex in the higher animals, and especially in apes and in man. In this connection it must also be borne in mind that there exist entire orders of birds (singing birds) with a completely regressive and unpaired bulbus olfactorius but whose regio entorhinalis is unusually well developed. Even parrots with a bulbus which shows still greater evidences of regression (although it is paired) have a demonstrable area corresponding to the regio entorhinalis. Reptiles with their well developed bulbus olfactorius and area prepyriformis have no regio entorhinalis (except *Varanus*, in which this region is small), whereas all reptiles have an extensive and well developed formatio ammonis.

Comparative anatomy shows that the regio entorhinalis may be entirely absent in animals with a well developed bulbus olfactorius, regio prepyriformis and cornu ammonis; on the other hand, there are some animals (singing birds, parrots) in which the regio entorhinalis is unusually well developed, although there is complete regression of the bulbus olfactorius and total absence of the cornu ammonis and regio prepyriformis.

These anatomic facts must undoubtedly be of great significance as far as the function of the regio entorhinalis is concerned. Nor can there be any doubt as to the physiologic significance of the morphologic fact that in microsmatic animals (apes, man) the regio entorhinalis occupies a considerable area and is subdivided into many subareas, with extensive differentiation of their elements. Just as important, perhaps, is the fact that the relative size of the entorhinal region, as compared with that of the bulbus olfactorius in lower animals, is in an inverse proportion to that in the higher animals, and especially to that in apes and in man. That is, the bulbus olfactorius is ever so much better developed in lower animals but much less developed in higher animals than the regio entorhinalis in either of them. The same relationship may also be said to exist, though perhaps not as marked, as regards the development of the cortex semiparietinus and bulbus olfactorius, on the one hand, and the regio entorhinalis on the other.

KESCHNER, New York.

PITUITARY TUMORS. OSKAR HIRSCH, Wien. klin. Wchnschr. **39**:93 (Jan.) 1926.

In this article the author discusses the ocular and acromegalic forms of pituitary tumor.

Ocular Form: This form of tumor is characterized by visual disturbances, without apparent change of habitus. There is, however, disturbance of the genital organs (impotence and amenorrhea), and close examination reveals symptoms that occur in pituitary tumors, such as falling out of the beard and the axillary and pubic hairs, disturbances of sweat glands, lethargy, carelessness and lowered temperature; but the most important symptom is the visual disorder, which is characterized by diminution of acuteness of vision and contraction of the visual field, the latter occurring in the form of bitemporal hemianopia. The author has studied his cases of pituitary tumors that have been verified by operation. In 84 per cent he found bitemporal hemianopia, the remainder being divided into central scotomas, homonymous hemianopias, nasal hemianopia of one eye and disturbances of the field of vision that were not characteristic.

The primary atrophy of the optic nerve, which occurred in 89 per cent of cases, was as characteristic as the temporal hemianopias. The remaining

11 per cent showed choked disk or optic neuritis. As a rule choked disk and especially optic neuritis suggest some other condition than pituitary tumor, and the author uses these symptoms as a means for diagnosing pituitary tumors only when they are accompanied by positive roentgenograms, genital disturbances, trophic disturbances, changed habitus, etc.

The reason that most pituitary tumors show only primary atrophy of the optic nerve is the fact that the tumor causes a cutting off of the optic nerve by way of the circle of Willis, and this barrier prevents the cerebral fluid from entering the sheaths of the optic nerve.

While changes in the eyegrounds frequently are noted in pituitary tumors, the tumors may occur with normal eyegrounds, especially in cases of short duration; this takes place because the cutting off is far back, so that a longer time must elapse before atrophy occurs.

Acromegalic Form: This disease was first described by Pierre Marie, in 1886, and since has been looked on as a homogeneous disease picture. The author, however, does not consider it as such and divides the thirty cases that he has observed into two groups—benign or classic acromegaly and malignant acromegaly. The pathologic-anatomic difference is the benignancy or malignancy of the tumor; the clinical difference is the visual disturbance. In the benign form, visual disturbances do not occur. In the malignant form, visual disturbances occur simultaneously with the development of the acromegaly, or later.

The visual disturbance not only tells whether a benign or malignant acromegaly is present, but also determines the location of the tumor. Visual disturbances occur only when the tumor grows out of the pituitary fossa over the level of the base of the skull. This corresponds with the anatomic topography of the chiasm and the pituitary fossa. The chiasm, as is usually supposed, does not lie close to the pituitary fossa in the chiasmal sulcus, but more than 1 cm. above and back of the chiasmal sulcus. The space between is occupied by the chiasmal cisterna, so that the chiasm itself is suspended in the cerebrospinal fluid. So long as the tumor does not extend over the level of the base of the skull, there are no visual disorders.

Benign Acromegaly: In this condition the tumor develops in the pituitary fossa and extends toward the sphenoidal sinus, with no tendency to extend to the base of the brain, and remains stationary. Hence compression of the chiasm and visual disturbance do not occur. Benign acromegaly usually (not always) is associated with a high grade hypertrichosis. Men have a hairy trunk, extremities and nates. Women are heavily bearded and are almost as hairy as the men. Genital disorders are less frequent in the benign form than in the malignant or other form of pituitary disease.

In this form of acromegaly especially, the parts of the body that are derivatives of the ectoderm show changes; in addition to hypertrichosis, the sweat and sebaceous glands are affected; there is thickening of the skin, enlargement of the tongue, defects of dental enamel, abnormal growth of nails, and one of the author's patients developed double cataract at 47 years of age. Severe headache is frequent, especially in the early stages. When the absence of headache in many cases showing a large pituitary tumor that has been present a long time is considered, the thought occurs that the headaches are not caused by a narrowing of the space brought about by the tumor, but are due to inner secretory processes, either a swelling of the brain or thickening of the skull cap, the former being the more likely since the headache promptly disappears after operation.

The benign as well as the malignant form causes great mental distress and should be looked on as a serious disease and not merely as a disease that mars the appearance. This form of acromegaly is caused by an adenoma in the anterior lobe of the pituitary which is composed of eosinophil cells. The adenoma emits a secretion, but as yet it is not certain whether the normal pituitary secretion is increased or whether it is changed in addition to being increased.

Malignant Acromegaly: This form is so named because the tumor continues to grow and is not stationary as in the benign form. However, even in the malignant form infiltration rarely occurs, and metastasis is rare.

The characteristic symptom of the malignant form is optic disturbance which is brought about by a gradually increasing tumor growth, which not only pushes the sella toward the sphenoidal sinus but also extends upward to the base of the brain. In this malignant form all characteristic symptoms of acromegaly are found: the masklike appearance of the face, the thick nose and eyelids, protruding lower lip, teeth far apart, thickening of tongue, etc. Contrary to the benign form, the hairy parts remain normal. Genital disorders are as constant as in other disease pictures caused by pituitary tumors—dystrophia adiposogenitalis and the ocular form. Headaches are not frequent as in the benign form, although often the tumor is larger.

The malignant type is explained on the basis that in the beginning there is an active adenoma producing a secretion which causes the acromegaly, but that the adenoma, if of malignant character, degenerates and loses its power to produce secretion and takes on the character of an inactive tumor such as is found in dystrophia adiposogenitalis and the ocular form. Therefore, the disease picture of the malignant form is acromegaly plus the symptoms of dystrophia adiposogenitalis and the ocular form.

The author classifies the types of pituitary disturbances from an historical standpoint as follows: (1) acromegaly (Pierre Marie, 1886); (2) degeneratio adiposogenitalis (Fröhlich, 1901); (3) die okuläre Form (Hirsch, 1911); (4) die hypophysäre Kachexie (Simmonds, 1914); (5) hypophysäre Diabetes insipidus (Simmonds, 1914); (6) hypophysärer Zwergwuchs (Erdheim, 1916); (7) dysostosis pituitaria (Schuller, 1921).

MOERSCH, Rochester, Minn.

THE RÔLE AND MECHANISM OF ACTION OF THE CEREBELLUM IN THE REGULATION OF MOVEMENTS. L. BARD, Rev. neurol. **1**:553, 1925.

It is nearly a century since Flourens localized in the cerebellum the faculty of coordinating and regulating movements. Since then clinical and experimental observations have multiplied, and an accord has been reached on this fundamental thesis. On the other hand, the interpretations of the mechanism of this action have been numerous and varied. Some have been merely wild guesses, but others have focussed until now a fair understanding has been reached. Investigations carried on during the last twenty years have brought Bard to a new conception of cerebellar function, the regulation of kinesias of opposite signs influencing protagonist and antagonist.

Granting that the peripheral mechanism is functionally intact, the initiation of the movement together with its nature and direction is the function of the cerebral cortex. Once this movement is initiated by the will, motor associations bring about the smooth performance of the act and determine the rôle of each of the neuromuscular elements that make up the whole. These mechanisms are automatic and subconscious, and are likened by the author to the pattern

of the roll that directs the playing of the mechanical piano. The seat of this function is located in the lower subcortical centers. These association centers mobilize muscles and parts of muscles and fix the degree of effort that each part shall take in the whole and the degrees of effort that one might speak of as differential, in contrast with the global effort set in motion by the cerebral cortex. The basal ganglia, mesencephalon, medulla, and spinal cord are the centers concerned in this ensemble.

Finally, there is a third process whose rôle seems to be the maintenance of the necessary collaboration of the protagonistic and antagonistic muscles. In the classic scheme the protagonistic muscles alone are active, and the antagonistic muscles, in order not to interfere with the contraction, elongate under the influence of reciprocal innervation and do not take any active part in controlling the movement. However, if it is the function of the protagonistic muscles to develop the force necessary to shorten the muscle and conquer external resistances and of the antagonistic muscles to transform this movement into a gradual harmonious whole, the author proposes to distinguish the negative kinesias or the active elongation of muscular fibers and the positive kinesias or shortening reaction. All muscles are called on to play this rôle of protagonist and antagonist alternately. They are innervated by the same centers. Therefore, these centers are themselves placed, turn by turn, under a charge of different sign. In this third system then, the center for regulating and correlating reflexes of positive and negative kinesias is the cerebellar apparatus.

In the cerebellar syndrome, the disturbance is found in the physiologic mechanism of muscular kinesias. In cases of cerebellar disease, such signs as clumsiness, incoordination, dysmetria, asynergia, discontinuity, adiadokokinesis, tremors, asthenia, atony, astasia, dysequilibrium, etc. are all the result of disorders in the kinesias. These disturbances are of three orders: (1) defect in proportion of positive and negative kinesias; (2) defects in synchronism, and (3) discordant variations, that is, the impulses launched are in direct opposition to those necessary for accomplishment. The exaggeration of amplitude results from the predominance of elongation of the antagonists over the shortening of the protagonists. Discontinuity results from faulty synchronization of the phenomena in respect to one another. The adiadokokinesis is caused by the difficulty of simultaneously reversing the signs of the kinesias. Asynergia results from defective synchronization in establishing the kinesias. The pendular reflex reveals anomalies in the arrest of the elementary impulse.

Visceral muscles do not take part in the cerebellar syndrome, because in them there is no interaction of contraction and elongation at the same time. That is, opposite phases in the visceral system occur in toto.

The essential difference that separates static from kinetic contractions is the fact that in the first case the two antagonistic groups are simultaneously in positive kinesis. This explains the immobility of the cerebellar patient in the attitude assumed voluntarily. Movement brings out defects in harmonious muscular contraction.

Another important function exercised by the cerebellum seems to be specific and deals with the sense of gyration. To be worthy of the term "special sense" there must be a physical apparatus differentiated to receive a particular physical phenomenon, a nerve that conducts its stimuli, a cortical center to perceive them, and a specific sensation to translate them. The sense of gyration seems to fulfil these postulates. The sensation of gyration deals only with the perception of movements of the head and has a vast effect on equilibration. Equilibrium results first from the motor associations giving rise to posture and,

second, from correcting reflexes of labyrinthine origin. Each displacement of the head immediately provokes a reflex that carries it in the opposite direction and brings it back to the initial position. This position is maintained by the alternation and equality of these reflexes in all directions. The rôle of the cerebellum in regulating equilibrium is the same as that by which it regulates all other movements. The pathologic cerebellar syndrome is constituted essentially by motor disorders that result from defects in the regulation of positive and negative muscular kinesias taking place during movement.

FREEMAN, Washington, D. C.

PUNCTURE WOUNDS OF THE CEREBELLUM. INJURY OF CEREBELLUM BY FOREIGN BODY ENTERING THROUGH JUGULAR FORAMEN, WITH REMOVAL AND RECOVERY. HAROLD L. FOSS, Arch. Surg. **13**:43 (July) 1926.

Foss reports a remarkable case of an injury to the cerebellum. A boy, aged 2½ years, was running with a double-ended ivory crochet hook in his mouth. He tripped, fell forward on his face and immediately began violently coughing and vomiting. In a moment or two he coughed out one-half the hook, which had broken at its center, and as the remainder could not be found it was supposed that it had been swallowed or insufflated. The child, although fully conscious, seemed totally unable to talk or cry. For a few minutes there was slight bleeding from the nose.

On examination, the respirations were slightly hurried and accompanied by a wheezing inspiratory stridor. The patient lay quietly, occasionally coughing and vomiting. There was a large quantity of mucus and vomitus in the mouth and throat which the patient seemed unable to expel. On its removal and on examination of the mouth and pharynx, lacerations were not disclosed. The missing crochet hook could not be found. General physical examination was negative. Fluoroscopic examination revealed the metal head of the hook in the upper pharynx, and on lateral view, it was found to project into the left cerebellar hemisphere for nearly its entire length. Stereoscopic plates were made, and the hook was localized definitely in the brain.

At operation a tiny punctured wound was found in the upper portion of the left anterior tonsillar pillar through which a probe could be passed for 5 or 6 cm., at which point it came in contact with the metal screw on the end of the crochet hook. It was necessary to incise the tract so as to admit forceps, several of which were tried before a satisfactory one was found. Finally, a long Hagar needle holder being used, the screw on the end of the ivory hook was grasped, and with a slight rotating motion the hook was removed. The object was firmly fixed, and a surprising amount of traction had to be used before it could be loosened. When it finally came away, there was a sudden and profuse hemorrhage from the wound, which was controlled by a long iodoform gauze pack. The child's respirations at once ceased. For ten minutes artificial respiration had to be resorted to, after which the patient began to breathe again; in a few minutes he was breathing normally.

When the patient returned from the operating room, the temperature was 97, pulse 120, respirations 50. That night he vomited slightly and coughed occasionally. Extreme restlessness was finally controlled by codein. On the following day the pack was removed. Twenty-four hours after the operation, the left eyelids became slightly swollen, and at the same time the left ear became discolored. During the first few days there were wide fluctuations in the temperature, but by the ninth day both temperature and pulse were normal. Following the operation and until the patient left the hospital thirteen days later, he experienced great difficulty

in swallowing, much of the liquid food returning through the nose. There were no urinary changes other than an increase in the specific gravity, and no glycosuria.

The child's recovery was normal. Changes in the eyegrounds indicating pressure were not seen at any time. Nystagmus or strabismus were never noted. Involvement of the facial nerve and changes in the reflexes did not occur. The child lay quietly in bed and, except for great difficulty in swallowing, seemed normal. There was difficulty in talking. He was out of bed on the fifth day. There was a tendency for him to fall to the left, and he could not walk a straight line. He was discharged on the thirteenth day. He still showed a slight tendency to veer to the left. He had some difficulty in swallowing for five or six weeks after returning home. The mother was obliged, therefore, to be cautious in selecting his food. Solids could be taken only with great difficulty, for on such occasions the patient would "choke up" and regurgitate froth and mucus. For several weeks speech was impaired, but it finally became normal. For a long time he talked only in a whisper. He was reexamined eight months after the injury, at which time he seemed normal in every particular.

A brief review of the literature on punctured wounds of the cerebellum accompanies this communication.

GRANT, Philadelphia.

PERNICIOUS ANEMIA, ACHYLIA GASTRICA AND COMBINED CORD DEGENERATION AND THEIR RELATIONSHIP. ROY R. GRINKER, Arch. Int. Med. **38**:292 (July) 1926.

The author discusses the association of gastric anacidity with changes in the spinal cord and the anemia of pernicious anemia. The absence of free hydrochloric acid in the gastric juice of patients having pernicious anemia was first discovered in 1866; since then, it has been determined that it is absent in a large percentage of cases of pernicious anemia. The author cites three of seventy-four cases in which free acid was found. One of these showed symptoms due to changes in the spinal cord. The most constant sign found and the one used by the author in making the diagnosis was the great variation in the size of the red cells, the average size being greater than normal. The achlorhydria of pernicious anemia is complete and unvariable, and remissions in the symptoms have no effect on it. It may be present long before the anemia develops. Gastro-intestinal symptoms usually precede hospitalization of the patient by some months.

Hurst believes that achlorhydria is a predisposing factor; intestinal infection takes place because the bactericidal action of the acid is lost. The infection then results in the absorption of two toxins—one is hemolytic and also acts on the bone marrow and the other is a neurotoxin and is the cause of degeneration of the spinal cord. Hurst found an intestinal infection with nonhemolytic streptococci in a large percentage of his cases. Kopoloff, on the other hand, found the same number of streptococcic infections in his controls. The author notes that infection of the gastro-intestinal tract is frequent in cases of anacidity and achylia gastrica.

McBride and Carmichael reject the gastro-intestinal theory and believe that the disease has an endocrine basis, because 96 per cent of their female patients had a myxedematous appearance and had ceased menstruating two years previously.

Lichtheim and Minnich first described subacute combined degeneration of the cord associated with pernicious anemia. At first the changes in the cord were supposed to be the result of the anemia, but since no such changes are

found in cases of secondary anemia, a theory that both the anemia and the changes in the cord were due to a toxin was developed. This toxin supposedly is composed of a neurotoxin and a hemolytic toxin. Either the anemia or the changes in the cord may never appear, in which case the toxin causing that symptom is supposedly in abeyance, though it is always potentially present. In some cases, in which a sudden anemia develops shortly before death, it has been noted that there has been a quick reversal from spastic to flaccid paralysis.

The percentage of patients having anemia who develop changes in the cord is variously stated by different authors as from 2 to 80. Grinker states that with high grade anemia paresthesias may develop without demonstrable changes in the cord; he therefore believes that numbness and tingling cannot be accepted as diagnostic of involvement of the central nervous system. He uses as his criterion definite objective neurologic signs. Of fifty-two patients who had paresthetic symptoms, only twenty-four developed combined degeneration of the cord. He states that 80 per cent of his patients who had paresthesia as the initial complaint developed degeneration of the cord, whereas only 20 per cent developed degeneration of the cord when paresthesia developed during the course of the disease.

The author concludes that combined degeneration of the cord may be caused by the hypothetic toxins producing pernicious anemia or developing during the course of the disease, but may also be the result of numerous other toxic conditions such as Addison's disease, carcinoma of the bowel, pellagra and arteriosclerosis.

WAGGONER, Philadelphia.

IODIZED SALT IN THE PREVENTION OF GOITER: IS IT A SAFE MEASURE FOR GENERAL USE? C. L. HARTSOCK, J. A. M. A. **86**:1334 (May 1) 1926.

Impressed by the great increase in the incidence of hyperthyroidism, and particularly by the appearance of an unusual type of thyroid hyperactivity in men during the last year, the author studied a series of these cases, with especial reference to etiology, and found evidence pointing to the continued ingestion of small amounts of iodine in the form of iodized salt as the primary factor. He presents a tabular analysis of sixteen cases of an unusual type of hyperthyroidism in man. He then calls attention to other groups of cases which fall in this category, although the symptoms do not differ from the usual ones: a group of eighteen women between the ages of 45 and 65 who presented no symptoms before using the salt and another group of thirty-two in whom the preexisting symptoms were greatly increased by the use of the salt. Another group was that of girls at puberty to whom, it had been thought, iodine could be administered freely; in five cases severe hyperthyroidism began after the use of the salt, and in numerous other cases mild nervous symptoms appeared after its use. Three other cases showed a return of symptoms four years after thyroidectomy, due to use of the salt. The author cautions against the postoperative use of iodine.

An interesting history of goiter treatment follows, which includes the work of Marine, Kimball, Boothby and Plummer and calls attention to several factors regarding iodized salt which must be taken into account when considering the work of these men. Believing with Phillips and Crile that unless the administration of iodine is properly regulated a fundamentally sound movement for the prevention of goiter may kill itself, the author reaches the following conclusions: (1) iodine should be administered to children from an early age

to puberty in small quantities; (2) careful examination should be made at puberty, and if an adenoma is present iodine should be administered only with great care if at all; (3) continuous use of iodine should never be prescribed for adults, and the pulse and weight should be carefully watched during periodic administration; (4) iodine should never be given to an adult with a fetal adenoma except just before operation; (5) adults with large colloid or colloid adenomatous goiters but without fetal adenoma should be treated cautiously; (6) iodine should be discontinued or greatly reduced when basal metabolism rate and symptoms improve in cases of exophthalmic goiter; (7) iodine should not be given in postoperative cases unless hypothyroidism exists, and then thyroid extract is preferable; (8) iodine is best administered in chocolate tablets containing 10 mg. of organic iodine or as Lugol's solution; periodic administration is preferable; (9) iodine may be given in very small doses to a pregnant woman to prevent fetal goiter; (10) hyperactivity of a thyroid gland induced by iodine does not subside on withdrawal of iodine, and operation is usually required.

The author's final conclusion is that the use of iodized salt, in particular, should be limited to periodic table use by children under the age of puberty.

CHAMBERS, Syracuse, N. Y.

THE COCOONING BEHAVIOR OF A SATURNIAN CATERPILLAR (DICTYOPLOCA JAPONICA); A PROBLEM IN ANALYSIS OF INSECT CONDUCT. NOBUMASA YAGI, J. Exper. Zool. **46**:245 (Oct. 5) 1926.

The author investigated the origin and significance of the so-called exit hole of the cocoon and the behavior of the larva of *Dictyoploca japonica* during and after cocoon formation. The structure of the cocoon is controlled by the surface character of the substratum with which it comes in contact. When the surface in contact was copper-wire net the caterpillar secreted the silk on the copper wire without derailment if the wire mesh was fine but divided the meshes into compartments with strands of silk if the meshes were large. When the surface was a glass wall, the portion of the cocoon in contact with it was a homogeneous carpet of silk threads. The width between the bases of the maxillary palpi and the irregularity in spinning, in the experiments with the copper-wire cage, on removal of the palpi led to the conclusion that the size of the meshes of the cocoon is controlled by the width between these palpi. By the reversal of the cocoon during spinning, the caterpillar's conduct was modified so as to fall into five different classes, according to the degree of exhaustion of the muscle tension of the mandibles and of the body and also of the material in the silk glands. The larva oriented itself upward, sometimes closing the lower opening and sometimes cutting a new opening upward with its mandibles or closing the lower opening without orienting upward. By double reversal of the cocoon during spinning, at the time of the first reversal, the insect made a new opening with the mandibles, closing the initial one at the under side, while in the second reversal it closed the new opening and the larva oriented upward without making a new exit. Three cases of reversal and one of double reversal of the cocoon were fatal to the insects. The formation of the exit hole at one end of the cocoon is more reasonably to be considered as a remnant of the larval habit so prevalent among case-making insect larvae. It is obvious that the formation of the opening is closely associated with the negative geotropism of the mature larva and that the so-called exit hole serves as such for the adult when the position of the cocoon remains unaltered during the entire period of its construction. The exit hole cannot

be a result of "foresight" on the part of the larva for the future benefit of the adult unknown to itself. Its formation is explicable on the basis of the behavior mechanism of the larvae and does not call for any anthropomorphic interpretation.

WYMAN, Boston.

STUDIES OF THE THERAPEUTIC EFFECT OF FEVER IN EXPERIMENTAL RABBIT SYPHILIS. JAY FRANK SCHAMBERG and ANNA M. RULE, Arch. Dermat. & Syph. **14**:243 (Sept.) 1926.

Some theories offered in explanation of the beneficial effect of malaria on syphilitic patients are: (1) a group relationship between the malarial and syphilitic parasite, immune bodies being formed by the malarial infection which inhibit the growth of the spirochetes; (2) shifting of the reaction between the spirochetes and the nervous tissue, the abnormal permeability of the meningeal and cerebral blood vessels permitting the ingress of catalytic agents; (3) broken down plasmodia serving as foreign proteins; (4) regeneration of blood cells following destruction which brings about a nonspecific stimulation of the defensive mechanism; (5) increased permeability of the small cerebral blood vessels which permits protective substances to reach the brain tissues; (6) pyrexia which burns up the toxins that affect the cerebral cells. It is interesting to note that in a large percentage of patients with general paralysis, who died with a high temperature or who had high temperature shortly before death, it is impossible to demonstrate spirochetes in the brain. Ordinarily, spirochetes cannot be demonstrated in the brain in about one third of the cases of general paralysis. It has been noted previously that high temperature has a beneficial effect on all manifestations of syphilis.

The authors have carried out a series of experiments on rabbits to determine the effect of elevation of temperature on the development of syphilis. The rabbits were inoculated with a syphilitic suspension and were then exposed to varying degrees of heat for varying periods of time by immersion in a water bath. In each series of experiments adequate controls were used, and in every case the control rabbits developed syphilis.

Spirochaeta pallida is apparently susceptible to destruction at a temperature far below that necessary to devitalize vegetable micro-organisms. In certain of the experiments the suspension of spirochetes was heated before inoculation, and although the spirochetes remained motile, the authors were unable to produce syphilis in the rabbit. They draw no conclusions as to the probable result of the application of similar conditions in man nor do they state that these experiments throw any light on the mode of action of malaria in syphilis.

WAGGONER, Philadelphia.

CEREBRAL DISORDERS FOLLOWING INJURIES TO THE CERVICAL SYMPATHETIC NERVES. NERI, Rev. neurol. **1**:417, 1926.

Weir Mitchell was the first to describe certain cerebral symptoms in wounds of the cervical sympathetic nerves. During the World War, a large number of cases of injury to the cervical sympathetic nerves was reported, but the attention of observers was concentrated on the analysis of this or that objective sign which experiment had produced in animals, without noting the effect on the individual as a whole. Neri observed nine cases of injury to the cervical sympathetic nerves. A striking feature in the history of these individuals was the severity of the phenomena that came on immediately after the injury. In this respect a response resembled more the response to cerebral injuries than

to injuries of the peripheral nerves. One man lost consciousness for about an hour; another became a prey to terrific anguish and, in spite of harsh commands to keep still he would not lie down, tore off his dressings, etc. The excitement lasted for three days. Headache is ordinarily intense, usually on the side of the injury and is pulsating, exaggerated by the least effort and by intense light. Sometimes scintillations, tinnitus and vertiginous attacks are present. These symptoms become attenuated after a period of time, but occasionally they recur with great violence. One man, seven years after injury, cannot remain in the sunlight without headache and vertigo developing. Another suffers hemicranic attacks and intense burning at the root of the nose, and this is followed by vomiting and sweating. Formication is occasionally present in the hand or even in the leg. Three patients developed attacks resembling epilepsy. Heated rooms and sunlight seem to be precipitating factors. Occasionally, consciousness is retained. The patients are irritable and easily fatigued, and association of ideas is slow.

Hyperemia of the nervous system suddenly produced by the section of the cervical sympathetic nerve is invoked to explain these disorders. Ophthalmoscopic examination at first shows congestion of the retinal vessels, and cerebral hyperemia runs parallel with this. The response to galvanic stimulation of the labyrinth is disordered. The symptoms may disappear if the sympathetic trunk regenerates. However, this has been observed only occasionally in these wounded patients. Most of the symptoms have remained stationary. Neri finishes with a warning that section of the cervical sympathetic nerve is not without danger.

FREEMAN, Washington, D. C.

SPASMOPHILIA OF ADULTS. G. PERITZ, Medico-Biological Journal, Moscow **2**:1, 1926.

In the course of investigating what becomes of spasmophilic children, the author discovered and described a new constitutional group of individuals presenting a definite relation, both etiologically and symptomatically, to the tetanoid condition of childhood. This he terms spasmophilia of adults. He includes under this heading the asthenic, slender individuals who have long narrow chests and relaxed abdominal walls with enteroptosis, abnormally long extremities relatively to the torso, pale skin and cyanotic, cold, moist hands. They present a great variety of symptoms including the various neuroses, migraine, epilepsy, angina pectoris vasomotoria, trigeminal neuralgia, sciatica, muscular atrophy, and even schizophrenia. The symptomatology of this array of conditions has for its common underlying cause an abnormal hyperexcitability of the peripheral nerves and of both the sympathetic and the vagus systems. The neuromuscular phenomena include unduly prompt and great response to electricity, Chvostek's sign, myoclonic contractions and hypertonus. Slight pressure over sensory nerves causes marked radiation of pain along distal distributions. The sympathetic overstimulation causes peripheral vasomotor angiospasm with good cardiac tone but with a normal or even subnormal blood pressure consequent on consistent dilatation of the vessels in the abdominal viscera. There is also an overactivity of the vagus system as evidenced by frequent occurrence of the Ashner reflex.

The condition resembles the vagatonia of Eppinger and Hess, from which it differs by the presence of sympathetic as well as vagus hyperexcitability. This conception is contrary to the pharmacologic bases on which the theory of Eppinger and Hess was founded. The author reconciles the difficulty by the belief that in the spasmophilia of adults the entire nervous system is in a state of hyperexcitability. This is dependent on endocrine imbalance, especially thyroid and

parathyroid dysfunctions, which cause metabolic disturbances, more particularly an alkalosis and a potassium-calcium deficiency. The author treats these cases successfully with appropriate glandular products and calcium preparations.

YASKIN, Philadelphia.

ON THE VESTIBULAR LABYRINTH OF THE ALBINO RAT AFTER PROLONGED ROTATION. H. V. GOULD, J. Comp. Neurol. **41**:223 (Aug.) 1926.

This study was done in collaboration with the Vestibular Committee of the National Research Council and consists of a report of a microscopic examination of eleven specimens of albino rats which had shown marked physiologic effects after they had lived for various lengths of time (usually from fertilization to maturity) in cages turned sixty or ninety revolutions per minute, some in clockwise and some in counterclockwise direction. Griffith and Detlefsen, who supplied these specimens, found that at the end of their rotation experiments these rats developed a permanent disturbance of equilibration, making circular movements about the nest, usually in the direction of the former rotation with the head twisted to one side. They concluded that these actions were caused by the rotation experiments.

Gould's report has an important contribution to offer on the interpretation to be made from these experiments when equilibratory disturbances follow rotation in the albino rat, for he finds that in all but two of the eleven rats under consideration there were injured labyrinths on one or both sides due to infections of the middle ear. Since it is the experience of others that infections of this sort are common among large colonies of albino rats, it is essential that data from experiments of this kind should be thoroughly checked against such existing conditions. In one case in particular, however, without any history of infection, a minute examination of the labyrinths, which had been exposed to rotations from fertilization to maturity, did not show any appreciable changes in form, position or orientation of any part of the equilibratory labyrinth when compared with that of normal rats. Gould's conclusions from observation of this specimen are that whatever behavioristic changes were induced by the experiment are either referable to cytologic changes not easily demonstrated, or to some effects on the central nervous system itself.

STONE, New Haven, Conn.

THE PSYCHOPATHOLOGY OF LYING. I. M. ALTSCHULER, J. Neurol. & Psychopath. **6**:20 (May) 1925.

Deception or lying in its very principle is merely a physiologic manifestation which finds its justification in the tendency to survive. In nature, mimicry, certain colors of flowers, the fox doubling his track, actions of birds when in danger, beetles shamming death, the web of a spider, etc., are instances of deception. The sense of weakness and fear are characteristic of animals practicing deception. In the matter of self-protection, primitive man did not differ much from animals. Without social organization man had no other standard of conduct than his own interest. Instinct and habit are the strongest factors in our nature. Pleasure, which is derived from exercising the function of instinct, must, in the process of lying, be great, because of its identification with self-preservation. A lie is more fascinating, more stimulating to the imagination. By deception, primitive man attained peace, safety and independence. Falsehood became a virtue with him. Internal processes contain an element of

deception. Nature takes "pleasure" into its service. It puts on the mask of pleasure to smuggle in the work of reproduction.

Children lie in spite of great efforts to teach them to be truthful. This is partly because they have much in common with lower animals. They feel a sense of inferiority. They lie to protect themselves. The memory of a child is far from perfect. Imagination varies inversely with age.

Man openly revolts against falsehood. The lie tends to be eradicated as an atavism. Reason cannot allow lies. The germ of deception still lives in man. The urge to lie becomes modified and harnessed—not eradicated. Art is beautified through deception. Fiction and poetry give us a deceitful satisfaction in living in a world of unreality. When the urge to lie exceeds its permissible degrees, it is called pathologic lying. This is only quantitatively different. It, too, serves the ego motive. The conscious lie is a wish, supported by subconscious impulse.

FAVILL, Chicago.

BLOOD SUGAR CURVES IN EPIDEMIC ENCEPHALITIS. J. KASANIN and G. P. GRABFIELD, Arch. Int. Med. **37**:102 (Jan.) 1926.

An increase in the sugar content of the cerebrospinal fluid in encephalitis has been noted by a number of observers. In the cases presented, the sugar metabolism was studied by means of the blood sugar curve. It has been shown that the sugar curve in encephalitis tends to be higher than normal and also that there is a quantitative relationship between the sugar content of the blood and of the spinal fluid in epidemic encephalitis. Thalheimer and Updegraff believe that there is a threshold level for the entrance of sugar into the spinal fluid similar to that maintained by the kidneys for sugar in the urine.

The authors studied twenty-four blood sugar curves in seventeen patients by the blood sugar tolerance test of Janney and Isaacson. The Wassermann reaction was negative in all cases. None of the patients had glycosuria. Four of the seventeen patients were in the acute febrile stage. The authors state that in none of the cases was there any question as to the correctness of the diagnosis. The change in blood sugar curve was so constant that it seems reasonable to conclude that there is a fundamental disturbance of the sugar metabolism in encephalitis, and also that the change in sugar metabolism persists in patients suffering from the mental sequelae of encephalitis. The authors thus conclude that this disturbance is associated with the underlying pathology of the disease.

WAGGONER, Philadelphia.

ON THE PHYSIOLOGY OF THE CENTRAL NERVOUS SYSTEM IN THE STARFISH, ASTERIAS TENUISPINA. A. E. HOPKINS, J. Exper. Zool. **46**:263 (Oct. 5) 1926.

That the central nervous system of the starfish, consisting of a circumoral nerve ring from which a radial nerve is given off to each arm, functions in the coordination of the activities of the animal as a whole is evident from the observations of many workers. Experiments were done on *Asterias tenuispina* to gain some concept of how one part of the nervous system may dominate all other parts and cause them to coordinate with it to produce locomotion in a particular direction. It was found that isolated arms of the starfish without connection with the nerve ring tend to move with their more basal ends leading, while those retaining connection with a part of the nerve ring tend to move with the distal ends in advance. This indicates a possible directive function of the nerve ring. It appears probable that each arm may be controlled by a

center in the nerve ring at the point of continuity with the radial nerve. Coordination of the arms may be so interfered with physiologically that two sets of arms result. The members of each group coordinate with each other, but the two groups are physiologically separate and pull against one another. It is probable that fission in the starfish is brought about in this manner. Section of the nerve ring at two opposite points causes the animal to divide in the plane determined by the wounds. The experiments indicate that the nerve-ring portion of the central nervous system of the starfish may consist of a series of physiologically separable though normally coordinating units.

WYMAN, Boston.

OBSESSIONS IN RELATION TO PSYCHOSES. ALFRED GORDON, Am. J. Psychiat. **5**:647 (April) 1926.

A number of cases are cited in which obsessions apparently constituted the first phenomena of a psychotic state and the basis for subsequent delusional development. Several patients had an early history of emotional instability with peculiar behavior and poor social adaptation. No attempt is made to evaluate the obsessive ideas and thus trace their relationship to the actual psychosis, although the author recognizes the advisability of such effort. Several patients committed suicide while depressed over their explanation of their unusual experience. A man, aged 35, a musician, began at 19 to complain of severe headaches, then of various somatic disorders. In spite of reassurances to the contrary he thought that there must be a physical cause for his depression and gradually began to fear for his mind. He then developed agoraphobia and other doubts which resulted in increased fear of losing his mind. Gradually ideas of reference appeared, together with those of persecution and poisoning, after which, within a few months expansive explanations followed and the patient died in a paranoid schizophrenic state. The author concludes that the direct transformation of obsessional ideas into delusional ones is possible but not frequent; but that they may constitute a point of departure for the development of delusions by a process of argumentative interpretation, which tendency is especially strong in paranoid individuals.

READ, Chicago.

TRYPARSAMIDE IN LATE NEUROSYPHILIS. J. ROSS MOORE and IRWIN C. SUTTON, J. Nerv. & Ment. Dis. **63**:569 (June) 1926.

The fact is beginning to be appreciated that arsphenamine, though strongly treponemicidal, destroys resistance and is at times even a disseminator of foci of the spirochetes. In late syphilis, when absolute sterility is impossible, it is of the greatest importance to build up resistance. Tryparsamide, because of its penetrability, tends to produce resolution and healing, and because of being less deadly to the organisms of syphilis seems to build up resistance in the host. The authors consider that bismuth, which is more spirocheticidal, makes an ideal combination with tryparsamide. Though small doses of tryparsamide, by stimulating subacute lesions of the optic nerves may cause some haziness of vision which if not stopped may lead to permanent atrophy, yet when discontinued early produce no more harm to the eyes than arsphenamine. The authors recommend careful ophthalmologic examination of the fundi and the fields of vision at the beginning and end of each course of injections of tryparsamide. When other drugs have failed in general paralysis its administration has caused improvement which is mostly clinical. In cases showing gastric crises and lightning pains the patients may be astonishingly improved.

Patients who have received intensive mercury and arsphenamine therapy beforehand show more striking gains than those without such preparatory treatment. Hence the greatest use of tryparsamide is in general paralysis and late tabes dorsalis and in the more resistive cases.

HART, Philadelphia.

MOLGULA CITRINA (ALDER AND HANCOCK). ACTIVITIES AND STRUCTURE OF THE FREE-SWIMMING LARVA. CASWELL GRAVE, J. Morphol. **42**:453 (Sept. 5) 1926.

The activities of the larva and the responses and orientations it makes to external stimulation, and the structural organization by which these activities are produced, are described and figured. The duration of the period of larval life is found to vary between five and 170 minutes. A proportionally small number retain the larval form a longer time. During larval life, periods of swimming movements alternate with periods of inactivity, the latter, at first of momentary duration, becoming longer and longer until activity ceases. A light receptor does not exist and response to light is not made. A statolith is found in the sensory vesicle, and frequent geonegative orientations are made during the free-swimming period. These are of short duration and tend to occur: when the larva emerges from the parent, at the beginning of each of the frequently recurring periods of swimming activity, and immediately on making contacts. Unoriented movements follow each orientation and constitute much the greater part of behavior. The larva lacks definite organs of attachment. The entire surface of the tunic becomes adhesive at the time of metamorphosis. Only those structures that make up the action systems of the larva are fully differentiated; all other parts are embryonic in condition.

WYMAN, Boston.

LYMPHORRHAGES IN THE MUSCLES IN EXOPHTHALMIC GOITER. LEONARD S. DUDGEON and A. L. URQUHART, Brain **49**:182 (June) 1926.

Nine cases of exophthalmic goiter (one associated with myasthenia gravis) were studied microscopically; the best results were obtained with the Giemsa stain. Lymphorrhages were found in eight of the nine cases and were both large and small; the large ones caused wide separation of the muscle fibers. The cell contents of the deposits were chiefly lymphocytes with some plasma and endothelial cells, and these were found in several instances in close proximity to small blood vessels. In addition to the lymphorrhages, proliferation of interstitial cells was frequently observed and at times there was a chronic interstitial myositis with resultant atrophy of the muscle fibers. In every case the eye muscles were more severely affected than the skeletal muscles, and the cardiac muscles were the least affected. The thymus gland showed nothing abnormal, while the thyroid presented the picture usually observed in exophthalmic goiter.

The pathologic symptoms in the muscles in myasthenia gravis referred to in the literature are exactly similar to those in exophthalmic goiter; the occasional association of the two diseases and the fact that enlargement of the thymus and certain ocular phenomena are common to both are cited to link together the two diseases. The authors have not seen such changes in the muscles in diseases other than those referred to.

STACK, Milwaukee.

THE EFFECT OF UTRAVIOLET RADIATION ON EXPERIMENTAL TETANY. W. W. SWINGLE and J. G. RHINHOLD, Am. J. Physiol. **75**:59 (Dec.) 1925.

The present study was suggested by previous clinical reports (Huldschinsky, K.: Deutsche med. Wchnschr. **45**:712, 1919; Sachs, F.: Jahrb. f. Kinderh. **93**:167, 1920) indicating that the ultraviolet ray is of value in infantile tetany.

Swingle and Rhinhold utilized a series of parathyroidectomized dogs fed on a tetany-producing diet. Prior to operation the animals were irradiated one hour a day for from four to twelve days, with a similar postoperative exposure continued until the time of death, which occurred in from two to twenty-five days. In all cases careful studies were made as to the blood calcium content. From analysis of the data obtained, it appears that the ultraviolet ray is apparently without specific effect on blood calcium level in either normal dogs or those operated on. Likewise it was found that, in spite of irradiation, tetany occurs in dogs operated on just as rapidly as in controls not operated on and not treated by irradiation and maintained on a tetany diet. It should be added, however, that in parathyroidectomized animals, although there was no evidence of actual prevention or cure, a definitely palliative effect was observed; life was prolonged and the more violent manifestations of tetany were much ameliorated. The authors feel that such benefit as is obtainable in tetany from the use of the ultraviolet ray depends on its effect in promoting a greater absorption and retention of food calcium with consequently a certain retardation in its rate of elimination from the organism.

RAPHAEL, Ann Arbor, Mich.

SOME OBSERVATIONS ON DEEP SENSIBILITY. O. M. DUTHIE, Brain **49**:187 (June) 1926.

The results of investigations by Head and Stopford on sensation, which like others were performed on patients known to have a lesion of some peripheral nerve, are reviewed. In order to study to what extent deep tissues are innervated by nerve fibers transmitted by the tendons, Duthie investigated twenty cases in which pure lesions of the tendons occurred between the wrist and the metacarpophalangeal joints. Sensation was studied by the methods of Head and Stopford with the following results: Tactile localization is not affected by the loss of these tendon fibers, and this form of sensation is not dependent on them. Recognition of passive movement and posture are affected only slightly, which suggests that nerve fibers associated with tendons are at most only slightly concerned with this form of sensation. Pressure-pain was found to respond to less pressure than the normal area; this is difficult to explain, but may be the result of disturbance of tactile pressure and not of pressure-pain. The author thinks that observations on pressure-pain presented some evidence that nerve fibers associated with tendons innervate chiefly those deep structures which are in the immediate neighborhood of the insertion of the tendon. There is not any definite evidence that fibers accompanying the long tendons are concerned with tactile discrimination.

STACK, Milwaukee.

TEMPERATURE AND RETINAL-PIGMENT MIGRATION IN THE EYES OF THE FROG. S. R. DETWILER and RUTH W. LEWIS, J. Comp. Neurol. **41**:153 (Aug.) 1926.

This paper is a continuation of studies of the retina of vertebrates previously published by Detwiler in the last few years. Miss Lewis, in collaboration with Dr. Detwiler, reports the movement of retinal pigment in a series of experiments on twenty-four light-adapted frogs subjected to darkness for either one or two hours at sixteen different temperatures ranging from 5 to 30 C., inclusive. Evidence of the results is summarized in one table and eighteen clear photomicrographs. The results agree with those obtained by previous investigators in the fact that at low and high temperatures in darkness the retinal pigment of the frog tends to assume the characteristic light position (expansion).

Marked contraction of pigment in previously light-adapted eyes will take place following one hour's exposure to darkness between 18 and 23 C., inclusive, which brings the intermediate temperature range for contraction greater than previous investigators have found. After two-hour exposures in the dark, the higher temperatures bring about less contraction of pigment than the lower ones.

STONE, New Haven, Conn.

THE COLLOIDAL PARAFFIN REACTION IN THE CEREBROSPINAL FLUID. A. MICHAEL CRITCHLEY, Brain **49**:226 (June) 1926.

One hundred and thirty cerebrospinal fluids were examined by the colloidal paraffin method according to a slight modification of the original technic of Kafka. All these tests were controlled by the colloidal gold test and readings were recorded as from 0 to 4, varying from slight precipitation of wax with no clearing of the fluid in the former to heavy precipitation of wax with complete clearing of the fluid in the latter. In cases not showing organic disease precipitation was not found. In tabes, the peak of the curve was reached in the fourth tube (11 tubes were used), while general paralytic fluids were characterized by complete precipitation in the first three or four tubes, the remaining dilutions rapidly becoming normal. Disseminated sclerosis likewise showed this type of curve and checked exactly with the colloidal gold test. Tuberculous meningitis gave abnormal curves with the peak of the curve, on an average, in the fifth tube. The author cites the following advantages of this technic over that of the colloidal gold; it is cheaper and simpler; the solution does not deteriorate so easily as gold; the actual readings are simpler; there is no variation in sensitiveness such as occurs in different batches of gold solution.

STACK, Milwaukee.

ASTHMA IN CHILDREN. II. THE INCIDENCE AND SIGNIFICANCE OF ECZEMA, URTICARIA AND ANGIONEUROTIC EDEMA. M. MURRAY PESHKIN, Am. J. Dis. Child. **32**:862 (Dec.) 1926.

This study was undertaken to determine the relation between angioneurotic edema, urticaria, eczema and asthma, indirectly from their incidence in a group of children with asthma. The material consisted of 100 children with asthma ranging in age from 6 months to 14 years. The children were tested not only with food proteins, but also with pollen, dust and bacterial proteins. Seventy-nine per cent were protein sensitive, and 2 per cent were nonprotein sensitive. Of the 100 cases, thirty-one presented a history of, or an existing, dermatosis, twenty-two of which were eczema, seven urticaria, and two angioneurotic edema. The author suggests that in young children with eczema, tests for sensitization might be utilized in the prevention of asthma.

VONDERAHE, Cincinnati.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Thursday, Dec. 16, 1926

DONALD GREGG, M.D., *President, in the Chair*

THE SYNDROME OF MENTAL AUTOMATISM AND ITS RÔLE IN THE PRODUCTION OF THE CHRONIC HALLUCINATORY PSYCHOSES. DR. PERCIVAL BAILEY.

The article contains a review of the views of de Clérambault, physician-in-chief of the Infirmerie spéciale de la Préfecture de Police de Paris. It will be published in full in the *Journal of Nervous and Mental Disease.*

DISCUSSION

DR. C. MACFIE CAMPBELL: Dr. Bailey has given an interesting glance into one chapter of psychiatry. French psychiatrists have, on the whole, tended to devote much time to description and to the analysis of clinical pictures with the use of somewhat general concepts such as that of degeneration. This method of approach has appeared to be less profitable than the systematic survey of the German psychiatrists, largely under the influence of Kraepelin, with much emphasis laid on the total evolution of the case. The influence of the freudian conceptions has developed another line of work. In view of this situation the work of the French psychiatrists has tended to be somewhat neglected, and it is of interest to have presented to us the conceptions of a modern French psychiatrist. Many of the views which have been presented might be open to considerable criticism but deserve scrutiny. It is not possible to come to a personal appreciation of the statements with regard to hallucinatory conditions without a systematic review of one's own material. The worker who stimulates one to review one's personal material from a new standpoint has done a considerable service.

DR. BAILEY: De Clérambault is not at all interested in the situation under which these hallucinations arise. His attitude would probably be the general French attitude that the situation is interesting for the understanding of the psychic content of the hallucinations, but has no dynamic power to cause them.

DR. DONALD GREGG: Would you explain on a toxic theory the often repeated story that a person just before drowning has a picture of his past life come into his consciousness?

DR. BAILEY: I never heard de Clérambault mention this phenomenon observed in drowning, but I imagine he might consider it as a toxic phenomenon.

DR. GREGG: I have recently been interested in watching a manic-depressive patient go through a manic attack. She said that she had found that her hallucinatory figures were motionless, and that in this way she could tell what was hallucinatory and what was real. It is not often that one finds a patient who will analyze hallucinations and try to differentiate the real from the unreal.

DR. BAILEY: De Clérambault's system has been built on the hypothesis that all the phenomena which he includes under the term "mental automatism" are of toxic origin, but this does not necessarily mean that he is a poor observer.

On the contrary, he follows the true French tradition in this regard; he is a keen, accurate and painstaking observer of clinical phenomena, and if he says that in a certain patient the psychosis began with echo of thought and other natural phenomena, and that the patient was not defiant or persecuted at the time, I should be inclined to believe him. He puts dementia praecox, paranoid praecox and the hallucinatory psychoses in the same group, all being on a toxic basis, their differences depending largely on the age of onset.

AN EXPERIMENTAL STUDY OF "HALLUCINATIONS." DR. HELGE LUNDHOLM.

Due to the courtesy of Dr. Morton Prince, an opportunity was obtained to make the following experiments on an interesting subject, Mrs. O., a former patient of Dr. Prince. The experiments were carried out in the new Department of Research in Abnormal Psychology at Harvard University.

Mrs. O. was formerly a "double personality," known as the case of the "Spanish actress," or "Maria Rosa." She is now well; the different integrates of personality cannot spontaneously come into dominance in her waking self any longer, but they can be experimentally reached in automatic writing, in automatic vision (hallucinations), the latter being the particular object of our study. The three "co-conscious" integrates of personality, which were investigated in their relation to the "hallucinations" were the ones Dr. Prince has called "Gracie," "Maria Rosa," and "the Work-shop."

These visions of Mrs. O. which with some hesitation have been called hallucinations seem to come spontaneously, independent of conscious thought on the part of the subject. Mrs. O. simply sits down in front of a gray board or any uniformly colored surface and, after a little while, she will begin to describe visions. The three questions we wanted answered by the experiments were the following: How vivid are these images? Are they due to a process of their own that can continue without Mrs. O. thinking directly of them; i. e., can they, in other words, continue and change while Mrs. O.'s attention is attracted to other activities? Where do they come from?

The observations bearing on the first question seem to indicate that the images approximate real perceptions in vividness and clearness. The experiments bearing on the second question did not give definite interpretable results. About the experiments bearing on the third question the following may be said: (1) Two somewhat different methods were used. Characteristics of both were that by the use of hypnosis and automatic writing mental processes were revealed going on co-consciously, while Mrs. O. was hallucinating. (2) In general, the experiments seem to confirm the results of a previous investigation carried out on the same subject by Dr. Prince. The essence of both Dr. Prince's observations and ours is that the hallucinations of Mrs. O. are closely related to thinking processes that go on outside her awareness—mental activity of co-conscious integrates of personality. (3) Furthermore, the nature of this relation seems to be this: The hallucinations appear to be translations into the language of moving images of the content of co-conscious thought. (4) The co-conscious integrate of personality that, in our experiments, was mostly responsible for the visions was "Maria Rosa," but both "the Work-shop and "Gracie" also were able to induce visions to the wakeful Mrs. O. "Maria Rosa" is furthermore able to induce visions to "Gracie," and may interfere with and modify visions induced by "Gracie" to the normal Mrs. O.

Concerning the wider significance of the experiments, I wish to express myself with caution. It may not be justifiable yet to draw any conclusions

from these concerning the nature of hallucinations in general. The experiments have first to be repeated on a larger number of subjects and particularly on subjects who do not present the clear-cut different integrates of personality that Mrs. O. does. But the indication of a definite mechanism of hallucinations, obtained even if only in her case, undoubtedly encourages further research and opens up a number of routes of inquiry in a field that is little explored. It forces on us a multitude of questions, each a psychologic problem of considerable interest: (1) Are the so-called eidetic images that have recently been discussed so frequently by psychologists of the same order as the hallucinations of Mrs. O., or are they different? (2) Are the hallucinations of a dementia praecox patient of the order of her visions or not? (3) Are the hallucinations of the dementia praecox patient, the delirious, and the confused patient all of the same kind and determined by the same mechanism that underlie the visions of Mrs. O., or are there among the insane different kinds of hallucinations determined by different mental mechanisms? (4) May some or other of the hallucinations of the insane be related to the eidetic images? (5) Are the auditory hallucinations of the insane analogous to the visual ones of Mrs. O. in this respect—that they are translations in the language of the perceived spoken word of thinking processes of which the patient is not aware?

Questions like these still await an answer.

DISCUSSION

DR. WILLIAM HEALY: I am interested in this subject from the standpoint of the criteria of judgment of eidetic types and of the question of voluntary imagery versus hallucinations. There has seemed to me all through the work of Jaensch and his fellows at Marburg a certain naiveté of judgment, a lack of attempt to develop any objective judgment; a reliance largely on the bare statement of children in regard to the possession of the eidetic type of imagery. And yet with this grave lack there come from the Marburg school proposals to reform education on the basis of whether or not the individual has eidetic imagery. Ten per cent of children are supposed to have powers of eidetic imagery, and these are to be taught on a different basis from the others. It is quite a question in this matter, and even in the work which Dr. Lundholm presents, of how much "playing the game" there is in the whole situation. How much response is there to suggestion? And how much does the child say about himself for fun or that he may be selected or noticed?

This even comes out in the question of hallucinations or illusions as against possible playing up for recognition by others. Which is which is sometimes difficult to determine. A boy under my observation, who was thought to be possibly developing a psychosis with hallucinations, made a play radio outfit of pasteboard and string and in his room at night he heard radio concerts and lectures coming in over his play machine. He barricaded his door and stayed awake long hours to enjoy these. Sometimes he told at great length the sermons, etc., he had heard. Everybody was fully persuaded he had hallucinations. Of course, it was a question of how much he was "playing the game" and how much there was of true hallucinations. The same question came out in the case of the chronic paranoics. I once revisited a patient after years and found that in the institution she had become the Queen of England. I saw her in her regal state and as I came into her room she said to me "You know that now I am the Queen of England, Doctor. You believe that, Doctor, don't you?" So one wonders how much there is of the real and how much

of the play element in such situations; how much there is of the creative imagination in the effort to produce certain effects. Just as in the case of the boy with the radio, this occurred in no less a person than Cardinal Newman, who tells frankly about developing hallucinations that commanded his moral life. They are reflected in his hymn "Lead Kindly Light"—"And with the morn those angel faces smile, which I have seen long since and lost awhile." These angel faces guided him distinctly over many years and were real to him during parts of his life.

Then one comes to a question of how much any of these things are due to unusual capacities for visualization. There are tremendous differences in the powers of visual memory. When I was interested in the psychology of testimony, I found many differences in the ability of people to see over again the things they had deliberately observed or even had not paid much attention to. Some people train themselves in the ability to see things over again. One of our best subjects was a scout leader who had trained himself to be observant with his boys. He was one of our best witnesses. One could say a great deal more on the whole topic, but this suffices to show something of the complications.

I should like to ask Dr. Lundholm if he gave calcium by way of experiment to see how it influenced eidetic imagery, as the Marburg school claims it does.

Dr. Lundholm: No. About dementia praecox patients, it is generally accepted that it is difficult to hypnotize them. I have never tried it myself. The risks that might follow such attempts, particularly the risk involved in allowing them to build up delusions of influence, have made me hesitate in trying them. It is therefore difficult to investigate the question whether or not the hallucinations of a dementia praecox patient are of the same order as those of Mrs. O. I feel doubtful that it can be proved definitely.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 17, 1926

W. G. Spiller, M.D., *President, in the Chair*

A Case of Left Hemiparesis with Aphasia and with Choreiform Movement in the Paralyzed Side. Dr. J. C. Yaskin.

This case is presented because of the unusual combination of a left hemiparesis with choreiform and athetoid movements and an aphasia in a right handed person. A negro merchant, aged 58, in whom, because of difficulty of speech and absence of close relatives, no satisfactory past medical history could be obtained, complained of weakness in the left arm and leg and inability to speak well. Preliminary examination in the dispensary showed a normal pulse and temperature, considerable cardiac enlargement downward and to the left, and a blood pressure of 200 systolic and 120 diastolic. Neurologically, there was narrowing of the left palpebral fissure, unequal pupils with the left smaller than the right, weakness of the left side of the lower part of the face, and distinct weakness of the left arm and leg with pronounced choreiform and athetoid movements. The tendon reflexes were distinctly increased on the left, but ankle clonus and the Babinski sign were not present. The patient was emotional and lachrymose.

The man was admitted to the hospital on the day of his presentation at the dispensary; shortly after admission he became drowsy and within two days comatose. He could be aroused, but was unable to make any sounds. He was unable to swallow and was incontinent. There was no spontaneous movement of the left arm and leg, but there was defense movement on stimulation. During this period of coma, the paretic limbs were flaccid, the tendon reflexes were increased as compared with the right side, but there was no clonus or Babinski sign. The choreiform and athetoid movements were now much less pronounced and during the period of coma practically disappeared. The blood Wassermann reaction was negative.

The patient gradually improved and four days after admission began to regain consciousness and control of the sphincters; he showed increase of power in the left arm and leg and, coincident with this improvement, there occurred a drop in the blood pressure to 138 systolic and 88 diastolic. Within three weeks he was up and about the ward. Both the left hemiparesis and the aphasia have continued to improve, up to the present time.

The temperature continued normal, and the blood pressure was 190 systolic, and 100 diastolic. Laboratory examinations revealed normal urine, blood count and blood chemistry. The roentgenogram of the head was negative. The spinal fluid in the latter part of November, 1926, showed four cells, slightly increased protein, negative Wassermann and colloidal gold reactions.

Examination in the early part of December, 1926, revealed the following: equal and regular pupils responding normally to light and in accommodation; impaired hearing in the left ear due to disease of the middle ear, and deviation of the tongue to the left with tremor. The visual fields were normal; the fundi were normal and showed no retinal sclerosis. The extra-ocular muscles were well innervated. Movements were possible in all joints, but the left arm was definitely weaker than the right in the arm, forearm and hand. Coordination tests were performed with precision on the right, but with considerable tremor and dysmetria on the left. Tests for adiadokokinesis were unsatisfactory, although there seemed to be some asynergia on the left. Quick jerking choreiform movements were seen occasionally in the left upper extremity; they were more pronounced on voluntary motion. There seemed to be occasional athetoid movements in the finger and thumb of the left hand; these, too, were more pronounced on voluntary motion. No rigidity or spasticity was noted on either side, and no fibrillary twitchings were seen. The left lower extremity was definitely weaker than the right. Extension and flexion of the knee and thigh and movement of the ankle were weak as compared with those on the right. The heel-to-knee test on the right was done with precision and on the left with some ataxia and dysmetria. The heel-to-shin test on the right showed no disturbance; on the left there was definite ataxia. Choreiform movements similar to those in the upper extremity were seen in the left lower extremity. Definite athetoid movements of all toes singly or in groups were evident. The tendon reflexes were more active on the left than on the right and the abdominal reflexes were present on both sides. There was no clonus or Babinski sign on either side. The Romberg sign was negative. The gait showed slight clumsiness with the left leg. The patient did not bend the left knee in walking; he dragged the leg slightly, swinging it out at the hip and coming down on the heel. The toes extended with each step on the left foot. There was no disturbance in vibration, touch or pain sensibility. The patient was perhaps hyperesthetic on the left side of the body.

The patient said that he had never learned to write, but that he used to cut and saw, and comb his hair with the right hand; it is therefore reasonable to assume that he is right handed. When examined two weeks ago, he could form words, but showed a definite tendency to a jargon type of speech. The sentences were short, often unfinished and lacked connectives. Many of his thoughts had no relation with one another and he was unable to give a clear account of anything. He failed to comprehend even a simple command such as to touch the nose with the finger. He could name some common coins but failed to name most of them. He considered a nickel worth more than a penny, and a dime worth more than a quarter. When told to select objects from a tray — a knife, scissors, match, key, etc. — he usually chose the wrong object and failed to name it when asked. Thus a knife was a "boilo," a scissors a "mistik," and a key a "kulte." He could tell, however, what the objects were for. Understanding of familiar imitative commands was poor. When asked to imitate the movements made by the examiner he always did the opposite of what was requested. In summary it may be said that the patient showed definite aphasia which was chiefly in the sensory sphere. Auditory and visual speech were alike affected, with the most marked failure in the auditory sphere. Even simple commands failed to be comprehended and writing was impossible, but the patient was illiterate. On the other hand, he could form words, though his sentences were not grammatical and were senseless. It should be stated that within the past two weeks there has been considerable improvement in speech.

Comment.—This case presents certain features of interest. There is definite aphasia, largely of sensory type, involving chiefly the auditory sphere, but also to some extent motor speech. In addition, there is left hemiparesis without rigidity and with choreiform and athetoid movements on the left side. The patient is right handed. The question arises as to what is the pathologic change and where is it localized. Lloyd and Winkelman reported before this society in January, 1924, a case of right hemiplegia with slight aphasia and acute choreiform movements on the nonparalyzed side. Necropsy showed thrombotic softening in the caudate nucleus, putamen, globus pallidus and knee of the internal capsule on the left side. On the right, explaining the choreiform movements, was a small area of softening of the inner margin of the putamen extending into the globus pallidus. The internal capsule was slightly involved. Arteriosclerosis was marked. Although the case of Lloyd and Winkelman differs from ours in that the choreiform movements were on the nonparalyzed side, it has much in common with this case and the pathologic change is probably similar. There is probably an arteriosclerosis at the basis of the condition. The aphasia is explainable by a lesion, probably thrombotic softening, in the left temporal lobe involving the superior temporal convolution. The choreiform and athetoid movements on the left together with the left hemiparesis and hyperesthesia can be explained by a similar area of softening in the right putamen with probably some involvement of the internal capsule. A bilateral lesion, therefore, on the basis of an arteriosclerosis would explain all that the patient shows.

DISCUSSION

Dr. Charles K. Mills: The probabilities are that this is a double lesion, which is by no means uncommon. Cases of pseudobulbar paralysis are often of this kind. The two lesions usually occur at the same time, and this is especially so in cases of encephalitis. The absolute flaccid paralysis is not uncommon in cases in which there is at the first a completed lesion.

TWO CASES OF TERMINAL PERIPHERAL NEURONITIS, RESEMBLING LANDRY'S PARALYSIS. DR. ROSS H. THOMPSON.

These cases show a rapidly progressive, symmetrical, ascending, flaccid paralysis of the extremities, muscular wasting and loss of tendon reflexes, with involvement of the cranial nerves and without implication of the spinal cord.

CASE 1.—In this patient, a woman, aged 48, the advance, within a period of two weeks, gravely involved the cranial motor functions to the level of the fifth nerve, also the vegetative fibers in the fifth nerve sheath, as shown by an ulcerative keratitis on the right and a lesser degree of keratitis on the left. Absent corneal reflexes indicated a sensory involvement; a papillitis of one diopter is unexplained except that it may occur in the wake of multiple neuritis.

CASE 2.—In this patient, a man, aged 27, apparently in good health, a similar advance to the same level, the fibers of the fifth motor nerve were less profound within a period of four weeks, there being no vegetative or sensory disturbance of that nerve.

The cases were contemporary; the disturbance ascended from the terminal fibers of the toes and feet and was predominantly motor in type; in both, processes unfolded in the direction directly opposite to the advance. In case 1 there was complete recovery within five months, with a return of all tendon reflexes except the Achilles; in case 2, within the same period of time, in the same unfolding manner, the man is well on the same road, having still some weakness and wasting in the extremities, particularly the lower, and absence of all tendon reflexes.

DISCUSSION

DR. T. H. WEISENBURG: The first patient I considered as having a typical case of the ascending type of Landry's paralysis, such as I saw during the epidemic of poliomyelitis in 1916. During the most crucial period, when breathing was most difficult and the pulse was erratic, I gave her directions not to move or make any effort; she obeyed and pulled through. In the beginning, Dr. John A. Kolmer prepared a serum from an old poliomyelitis patient; this was injected intraspinally three times and may have been a factor in the recovery. Besides the bulbar symptoms mentioned by Dr. Thompson, this patient had a papillitis of about 1 diopter with keratitis on one side. I have never seen such complications in poliomyelitis nor are they mentioned in the literature, so far as I know.

The second patient is somewhat different and resembles the cases reported by Dr. Kennedy under the title of neuronitis. This patient has atrophy, fibrillary tremors and loss of power, everything to indicate involvement of the peripheral neuron. Both patients had pain, but that is usual. In fact, if any patient with a presumptive diagnosis of acute poliomyelitis did not have pain, I would not make that diagnosis.

DR. CHARLES POTTS: I understood Dr. Weisenburg to say that the patient at one time had fibrillary twitching of the muscles. This interests me because I saw a patient several times recently who had gradual involvement of the limbs —weakness first in the legs and then in the arms, with numbness of the feet and hands—muscular atrophy, no typical action of degeneration, fibrillary twitching in the muscles of the hands and loss of vibratory and position sense of the toes. On account of the sensory symptoms, I did not think it a case of true spinal muscular atrophy. The patient has been improving. I looked on it as a case of possible neuronitis.

Dr. F. X. Dercum: The fact that there have been sensory symptoms in the present case would be in favor of the involvement of the nerve trunk. In regard to the first case, it seems to me there was abundant cause for infection. There was involvement of the mucous membranes of the air passages for a long time. One often forgets a passing cold and thinks that it is not worth mentioning, but just such a cold may be the cause of the disorder.

Dr. W. G. Spiller: Does any member care to discuss the difference between neuritis and neuronitis? I should like to know what distinctions are to be held. I know that Dr. Kennedy does make distinctions, but what are the distinctions?

Dr. F. X. Dercum: As the parts involved are parts of one structure, the nerve cell and its axon, i. e., the neuron, I think the distinction must be difficult to make. Perhaps the presence of fibrillary tremors would be in favor of a primary lesion of the nerve cell rather than of the motor fiber. If neuritis is not purely motor but involves other fibers as well, sensory phenomena, trophic changes in the skin, changes in the oil or sweat glands, would be in favor of neuritis. Such changes could not be present in a parenchymatous neuritis limited to the motor fibers. On the whole I regard the differentiation between a primary disease of the motor nerve cell and of the motor fiber as difficult and doubtful.

Dr. Charles K. Mills: Neuronitis is a correct term. This is a condition in which not only the peripheral nerves but also the central origins of these nerves are involved and I cannot see any reason why, from an infection, both these parts may not be involved. A number of cases of neuronitis were reported during the war, and other military cases have been reported since the war. The subject was brought up in this society some years ago and the term neuronitis was then indorsed.

Dr. Ross H. Thompson: Kraus describes three types of peripheral neuronitis; the terminology used does not aim to discard older terms but merely to group them. Each presents different clinical features: (1) cellular peripheral neuronitis, in which group are poliomyelitis, herpes zoster and Landry's disease; (2) truncular peripheral neuronitis, which includes radiculitis, cauda equina disease and injuries to nerve trunks; (3) terminal peripheral neuronitis, involving the distal terminals of nerves, a large number of which are usually grouped as multiple symmetrical neuritis, polyneuritis, etc. The two cases presented would fall in the third category.

Two Cases of Friedreich's Ataxia with Especial Reference to the Rôle of Alcoholism. Dr. P. E. Kubitschek.

Two cases of Friedreich's ataxia were shown. The patients were brothers, the third and fourth children in a family of six, of which the first child died at the age of 14 months of whooping cough. The second child is a boy, now 16 years of age and in excellent health. The third and fourth children are the patients presented; the fifth is a boy, aged 5 years, in good health, as is also the sixth child, a girl of 3 years. The various theories concerning the nature of this disease were reviewed, with especial reference to the factors of etiologic significance. The rôle of alcoholism in the parents was stressed and the literature bearing on this phase was reviewed.

Case 1.—A boy, aged 14, of normal birth and development up to the age of 11 years, had had no serious illnesses or injuries. At the age of 11, slight clumsiness in walking and running with an increasing tendency to stumble

were noticed. A feeling of tiredness after walking for any length of time and on standing soon followed; the condition had steadily progressed to the present staggering gait and definite weakness of the back and legs. The knee and ankle reflexes were lost; the tendon reflexes of the arms were diminished; the skin reflexes were active; there was a typical Friedreich's toe, with bilateral Babinski reflex, slight scoliosis of the spine and marked incoordination of both upper and lower extremities. The roentgenogram showed spina bifida of the fifth lumbar vertebra.

CASE 2.—The birth and early development of this patient were normal. At the age of 10, gradual loss of weight was noted, but there was no evidence of illness. A few months later a tendency to stumble developed; this rapidly progressed and was accompanied by moderate weakness. The ataxia soon extended to the upper extremities and muscles of the neck. A year after the onset a change in speech was observed. At present there is advanced ataxia involving the trunk, arms, legs and muscles of the neck with speech disturbance, scoliosis, tendon reflexes diminished in the upper extremities and lost in the lower, positive Babinski sign and typical Friedreich's toe, slight impairment of sense of position and vibration in both feet. The roentgenogram of the spine is negative. Mental impairment was not seen in either of these cases.

In the family history, extending back over four generations, large and moderate families predominated; there was no history of similar illness in either the maternal or the paternal lines, neither was there any history of alcoholism or other excesses, except in the case of the father. The history of the mother was entirely normal; she was 21 at the time of marriage. The father was 22 at the time of marriage, had been temperate previous to that time and remained so until the time of the birth of the second child. At that time he suddenly began to drink heavily and for a period of five years took from fifteen to twenty glasses of whisky daily. During this period three pregnancies occurred, the first and second resulting in the birth of the two patients, the third resulting in a miscarriage at six months. After the miscarriage, the husband stopped drinking almost as abruptly as he began. Two pregnancies after the alcoholic period have resulted in the birth of two healthy and apparently normal children, although they are not yet old enough to justify the conclusion that they will not be subject to the disease.

The conclusion drawn from this study was that severe alcoholism, in a case in which neither personal nor hereditary tolerance exists, may have an injurious effect on the germ plasm and in this manner act as an exciting factor in the production of this disease.

DISCUSSION

DR. J. H. LLOYD: Friedreich's ataxia in two members of the same family is considered, and no doubt is, rare. Nevertheless, I have seen three instances. A brother and sister with this disease were in the Philadelphia General Hospital for a long time. A few years ago I had two colored boys, brothers, about 11 and 8 years of age, under my care with this disease, and I showed them before this society. They were typical examples, the swaying movements being characteristic. Friedreich's ataxia must be rare in the negro race. The elder boy died of influenzal pneumonia four months after he was admitted to the hospital. The cord was examined by Dr. Newcomer, and showed typical changes. These changes were gliomatosis. They were not confined to the posterior columns but were more widely spread, invading the lateral regions and especially the cells in Clarke's column and the direct cerebellar tracts.

I recollect a case of typical Freidreich's ataxia, with Babinski reflexes, showing that the lateral tracts were involved.

These cases in brothers raise an interesting biologic question, for this disease is apparently caused by some developmental defect. The colored boys were two of five children, the other three of whom did not show any symptoms. The parents were healthy. There was no syphilis in the family. It was thought at first they might be cases of juvenile tabes. Tests were made of the spinal fluid, and blood tests of the mother and older sister were made; they were all negative.

Friedreich's ataxia may have some kinship with Marie's cerebellar ataxia and even with syringomyelia. In all these diseases there is a gliomatosis. Gowers proposed to call this underlying condition abiotrophy, but this is merely giving a different name to it and does not explain its nature or causation. There is apparently some defect in the development of the nervous system, but just what this is constitutes the problem.

Dr. Charles Potts: A brother and sister suffering from this disease were for a long time in the Philadelphia Hospital. The sister is still there; the brother died some years ago and I think was reported.

Dr. F. X. Dercum: Was a roentgenogram taken of the thymus gland?

Dr. P. E. Kubitschek: No.

Dr. Raymond Waggoner: The cases mentioned by Dr. Patten were presented by Dr. Weisenburg. As I remember, there were five in the family who showed the typical Friedreich's ataxia; of the five, two were boys and three were girls. Two of the girls were in the hospital for some time. I do not think that all the members of the family who showed Friedreich's ataxia had spinal bifida, but two or three other members of the family also had a spinal defect.

The Association of Multiple Neuritis with Syphilis. Dr. W. B. Cadwalader.

The occurrence of multiple neuritis from syphilis has been under dispute for a long time. One of the most recent writers to consider the subject is Petren (abstract in *Progressive Medicine*, September 1920, p. 344), who came to the following conclusions: "1. Syphilis may cause multiple neuritis during the first year after infection, but such an occurrence is very rare and there are only eleven cases which may be accepted as incidences of this type. 2. It has not been demonstrated that syphilis of a duration of more than one year causes multiple neuritis. 3. Acute mercurial intoxication may be the cause of multiple neuritis, and probably chronic mercurial intoxication may also be a cause."

Souques has called attention to the loss of the Achilles tendon reflexes in syphilitic persons during the course of treatment with arsenic preparations. Schamberg and Greenbaum (*Progressive Medicine*, September, 1925, p. 336) reported a case in which after four injections of neoarsphenamine there appeared all the characteristic symptoms of multiple neuritis, with severe paralysis of all four limbs. The man was bedridden; tendon reflexes had disappeared and there was tenderness over the nerve trunks. I have seen a few cases of syphilis in which the subjective symptoms of numbness, tingling, paresthesia, pain, etc., had developed in the extremities after the administration of the arsenic preparation, suggesting, therefore, an arsenical form of multiple neuritis.

A man, aged 48, is known to have syphilis, with positive Wassermann reactions, but is apparently in perfect health and without symptoms. After

an exposure to cold and wet in September, 1926, he developed a respiratory infection followed by weakness of all four limbs, paresthesia and tenderness of the muscles, bilateral foot drop, and bilateral wrist drop. It required about ten days for these symptoms to develop. He was treated with mercury while in the hospital. The symptoms gradually subsided, and he recovered the use of the limbs; at present he is nearly well. He still has subjective sensations, paresthesias, etc., in the limbs, with some tenderness over the nerve trunks, and loss of the patellar and Achilles tendon reflexes. All other symptoms have disappeared.

In my opinion, the symptoms of multiple neuritis were caused by the infection, and the association of syphilis was accidental.

THE VASOMOTOR AND PILOMOTOR MANIFESTATIONS IN TUMORS AND LESIONS OF THE SPINAL CORD. DR. TEMPLE FAY.

This paper will be published in full in a later issue of the ARCHIVES.

PAROXYSMAL SPASMS OF THE EYELIDS AS A POSTENCEPHALITIC MANIFESTATION. DRS. B. J. ALPERS and C. A. PATTEN.

This paper will be published in full in a later issue of the ARCHIVES.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 4, 1927

I. ABRAHAMSON, M.D., *President, in the Chair*

BRACHIAL PLEXUS PARALYSES. DR. VINCENT GILIBERTI.

Brachial plexus paralyses form the second largest group of birth palsies. They occur most often in cases requiring artificial aid in parturition and are more frequent in head than in breech, shoulder or foot presentations. The mechanism responsible is traction on the plexus due to abduction, elevation and backward movement of the right arm at the same time that the head is bent toward the opposite side. Compression or stretching of the brachial plexus in attempting to free an arm or deliver a shoulder or after-coming head, will also produce similar injuries. Introduction of a finger or hook into the axilla when the shoulder is delayed may lead to compression, either by direct pressure on the plexus or indirectly by pressure of the clavicle, when the shoulder and clavicle are pushed backward and upward. Forceps blades rarely may reach the cervical region and damage the plexus. More often this form of paralysis is caused by pressure or traction exerted on the shoulders in attempting to accelerate the birth of the head by such methods as the Prague, Smellie-Veit or Mauriceau, or the combined methods. In rare instances, compression of the plexus has been caused by twisting the umbilical cord around the neck. Complications of obstetric paralysis may be fracture of the humerus or clavicle, dislocation of a shoulder, hematoma of the sternocleidomastoid muscle and separation of the epiphyses. These complications are no longer looked on as the actual cause of the paralysis.

The nerve fibers and bundles of the plexus are usually torn apart and not merely crushed or cut in one place, as in most traumatic nerve lesions. Overstretching breaks the funiculi at any point from the spinal cord origin of the

roots to the most distal point of trauma, causing irregular symptoms. Examination of cross sections of the resulting scar discloses incomplete interruption of continuity. It is difficult for a surgeon to determine exactly at what level excision of the scar will give him intact funiculi, for there may be interruption in a number of places.

Lesions of compression, traction or laceration vary only in degree. After slight compression, restoration of function is usually rapid and complete. If greater compression operates over a comparatively long time there may be radical organic changes resulting in disintegration of the fiber extending the entire length of the nerves distal to the site of injury. If traction has been the cause of paralysis, the disturbance may be transient or permanent depending on the degree of trauma. In cases of avulsion of the root from the cord, the cord has been found smaller and flattened in its anterior portions at the level of the injury, and the anterior horn is involved in a sclerotic process. The gray matter is deformed on both sides; the cells are completely absent on the affected side, and on the opposite side only a few darkly stained elongated forms remain. The most common type of brachial plexus paralysis is the upper arm type (Duchenne-Erb), due to injury of the fifth and sixth cervical roots or the upper primary cord of the plexus which originates from these roots. The lower arm type (Klumpke-Dejerine) is caused by a lesion involving chiefly the seventh and eighth cervical and first thoracic roots. This form is often the residual of a combined type; sensory symptoms are usually present, but, as in the case of Erb's palsy, are difficult to elicit in infants. The combined type is caused by a lesion involving the entire plexus or most of it.

The prognosis of obstetric brachial plexus paralysis is much less favorable than that of ordinary peripheral nerve lesions. Recovery from mild compression traction paralysis may take place in a few days or weeks. If severe, it may take as long as two or three years. If actual laceration with severance of continuity, separation and displacement has taken place, one can expect recovery only by resorting to surgery. From the standpoint of clinical types, the upper arm type offers the most favorable prognosis, the lower arm type next, and last the combined type. With long existent obstetric paralysis a habit palsy may replace the organic one when nerve conduction is finally restored.

The various complications should be treated surgically. The paralysis is taken care of by immediate immobilization of the affected extremity to prevent additional hemorrhage and further separation of nerve segments. A splint, extending from hip to axilla, with the arm elevated in abduction at an angle slightly more than 90 degrees, will help to approximate the torn nerve ends. The hand and forearm should be in full supination to overcome the tendency to pronation. The general nutrition can be favorably influenced by electric stimulation, massage, heat, hydrotherapy, and exercises, which should not interfere with the postural treatment. If signs of regeneration have appeared within one year, the mechanical and physical treatments should be continued. If there are evidences of partial regeneration or no regeneration at all after a year, one is justified in resorting to surgery. Contractures are best overcome by the method of gradual correction.

DISCUSSION

DR. T. P. PROUT: About twenty years ago, Taylor, L. P. Clark and I presented about six cases, mainly of the type induced by rupture of the outer cord of the brachial plexus. In several of these the lesion was in such position that excision accomplished a fair result; in others the lesion was more complicated

and the result was less favorable, but some improvement was shown. In all the condition had existed for a long time, so that any improvement was a gain. There were no early lesions in that series. Subsequently, I saw several early cases at the Vanderbilt Clinic; some of the patients did well with massage. The lesion is easy to palpate in new-born infants and in younger children. Even neglected cases are not hopeless. Excision of the lesion and resection of the nerve ends accomplishes something.

Dr. John Hugh Nolan: The time to help these palsies is near the time of their occurrence; the sooner they are treated, the easier it is to get results. As Dr. Giliberti said, it is the degree of injury that counts. During labor it is sometimes necessary to exert great traction, so much that at times one can actually hear the nerve trunk snap. I have followed many of these palsies; any improvement usually occurs within the first six months. I have seen a few infants get well, but they had minor injuries. In the severe types, the chances of good recovery are poor.

Dr. Michael Osnato: Dr. Giliberti did not have the opportunity to speak about the clinical aspect of his case, which illustrated several points he made in his paper. I have seen the girl several times. She did not improve perceptibly for a long period, and then, rather suddenly following an infection, she made what appeared to the family to be an almost miraculous recovery in a few months. When seen at the New York Post-Graduate Clinic, there was no reaction of degeneration, and in spite of some wasting in the shoulder girdle group the reflexes were normal. There were few, if any, residual sensory disturbances, and apparently what had happened was that she had developed a habit paralysis; when the habit disappeared, recovery, which had unquesitonably taken place a number of years before, was allowed to become evident. It might be well to stress again the point that surgery is not the method of choice, so far as routine treatment is concerned, in these cases. The postural treatment with prolonged physiotherapy is the regimen which offers the best results.

Dr. Charles Rosenheck: Many of these patients drift to an orthopedic institution. It is difficult to take electrical reactions in children; one should try to determine the amount of palsy by the position of the arm and the amount of functional disuse. Orthopedists put the arm in what they call the "aeroplane" position, i. e., abduction to the horizontal in an externally rotated position. The infants usually do not object to this posture, but the mothers do, because it entails difficulties in dressing and undressing the child. The results are good when nerve avulsion or tearing is not present. The treatment is continued for from four to six months. The infant is then brought in for massage and electric treatment. This is kept up for from six months to two years, and if recovery does not take place, the orthopedist then undertakes operative procedures (devised by Dever of Boston), dividing those muscles which keep the arm fixed in an adducted and inwardly rotated position. After this operation, the arm is again put up in the "aeroplane" position, in which it is kept for possibly two or three months; the electric treatment, exercises and massage are continued. The operation prevents awkward inward rotation and offers a fair amount of functional recovery.

Dr. I. Abrahamson: I wish to stress two points in the examination of these children: (1) a record of the movements that are preserved should be kept; (2) in order to discover the movements possible, the child should be tested by noting the resistance to passive movements. The child will resent passive movements, and one group after another can be tested in this manner.

Dr. Giliberti: The patient who was to have been presented this evening was 14 years of age when she came to the hospital, and had absolutely no use of the right arm. There was a generalized atrophy from disuse affecting the entire upper extremity. She had become accustomed to writing with the left hand, and at the same time had a peculiar sensory disturbance over the radial side of the lower half of the anterior surface of the right forearm, which one might think had something to do with the brachial paralysis. She gave a definite history of a brachial paralysis. A patch of atrophied skin, however, led to a diagnosis of morphea, and it was considered that the anesthesia and other sensory disturbances were caused by this. Under arsenic treatment, the skin lesion improved and with it the sensory disturbances. In about two and a half months, the patient could use the extremity normally. Within six months the handwriting was legible, and when I last saw her, about a year after treatment was begun, she wrote better with the right hand than with the left. I believe this was really a case of habit paralysis.

Adhesive Spinal Arachnoiditis Simulating Spinal Cord Tumor. Operation and Complete Recovery. Dr. Byron Stookey.

This aricle appeared in full in the *Archives of Neurology and Psychiatry* **17**:151 (Feb.) 1927.

DISCUSSION

Dr. Abrahamson: Does one see, in these cases, an acute exacerbation of symptoms following lumbar puncture, such as one occasionally observes in tumors of the cord?

Dr. S. P. Goodhart: We are all familiar with Dr. Stookey's contributions on this subject. The clinical forms which he has described are becoming more familiar as one recognizes the symptoms and associates them with this form of lesion of the spinal meninges. The case presented differs from most of the cases heretofore described, as I recall them, in that it presents a more acute symptomatology; the case doubtless belongs in the category of those previously described, but differs from them in that the duration is longer. This presentation brings to my mind a case of arachnoiditis, seen at the Bronx Hospital, which ran an acute course. Having seen several cases of this group, I was able definitely to make the diagnosis. Dr. Joyner, at my suggestion, operated and found manifest inflammatory arachnoiditis with adhesions and pressure on the cord. A diagnosis of encephalitis had previously been made. The patient was brought into the hospital, however, as a case of pneumonia. When I first saw her, there were: definite hyperalgesia at the level of the third and fourth thoracic dermatomes; a definite Kernig sign, rigidity of the neck, and nystagmus. The spinal fluid was at first under great pressure; it contained only a few cells, with slight increase in globulin. About ten days after the acute onset, mild tonic fits occurred, with periods of unconsciousness, sudden general rigidity and dilatation of the pupils. Subsequent spinal punctures showed gradual diminution of pressure and within a few days definite xanthochromia, with 300 polymorphonuclear cells and marked increase in globulin. The area of hyperalgesia remained definite. There were no sensory changes and no definite pyramidal tract signs. The sugar disappeared from the fluid. The picture strongly suggested to me the type of localized lesion with an acute onset, which Dr. Stookey has described; it is of interest to note that the fluid pressure became gradually so reduced as essentially to disappear. There were no organisms in the fluid. I felt that there was definite evidence of a localized lesion, inflammatory rather than neoplastic. The operation confirmed the suspected condition.

I feel that some of the cases which simulate cranial neoplasms, especially at the base, and in which operation fails to reveal a growth, are really of the character described by Dr. Stookey.

DR. E. D. FRIEDMAN: Was there evidence of formation of an arachnoid cyst in this case?

DR. HENRY A. RILEY: I have been greatly interested in this condition. At the Neurological Institute, I have seen three or four instances, and the matter has come up at conferences. Two conflicting points of view particularly struck us during their consideration. Most of the cases presented rather a long course leading up to the picture that simulated a neoplasm of the spinal cord. There usually is definite interference with the long conducting tracts, both those coming toward and going from the brain, with pyramidal tract signs, more or less indefinite sensory level symptoms, sphincteric disturbance and a certain amount of pain. When lumbar puncture was done, they presented the classic appearance of a tumor of the spinal cord. On the one hand there was the incomplete clinical picture of a tumor of the spinal cord, and from the special investigative point of view, there was the classic picture of a tumor; it is the balancing of the two features that is important in determining whether the case is one of arachnoiditis adhesiva spinalis, or a tumor of the spinal cord. All patients who have even indistinct evidence of a level lesion with interference with the circulation of the cerebrospinal fluid should be operated on; if this condition is found, they can often look forward to an amelioration of the disease, if not to complete recovery.

DR. WILDER PENFIELD: It is of the utmost importance to bear in mind the point that Dr. Stookey has made—that a complete block may exist without any xanthochromia or increase in protein content. This case is typical in several respects: the absence of xanthochromia; the slow development of symptoms, and a well localized and well circumscribed arachnoiditis. This might bear out the point of view that the increase in protein below the tumor block is caused, not by exudation from the vessels of a tumor, but by localized constriction of the vessels in the spinal cord. That is also borne out by the fact that xanthochromia develops even when the tumor is a chondroma which has almost no blood supply, and when the tumor is outside the dura, and in cases in which the compression is from a cold abscess. It seems to me that this type of well circumscribed arachnoiditis, with rapidly developing symptoms, may well compress the veins in the cord itself and thus give rise to increase in the protein; Dr. Stookey has pointed out in the other cases, in which the adhesions are more diffuse, that there is not any increase.

DR. ROSENHECK: Can Dr. Stookey explain the absence of spinal arachnoiditis in cases of meningitis in childhood that are followed for a great many years and do not develop a spinal fluid block? Possibly his suggestion that the ordinary infectious processes produced circumscribed serous meningitis may not apply to the meningitis of the meningococcus variety.

DR. STOOKEY: In answer to the chairman's question as to whether the symptoms were increased after lumbar punctures, I do not recall that I ever saw a case in which they were. I have seen one or two cases in which the symptoms were improved, but I would not be willing to say offhand whether any were made worse.

As to Dr. Goodhart's comment, I would recall to him the cases described by Dr. Horrax of adhesive arachnoiditis of the posterior fossa which simulated cerebellar tumors.

Dr. Riley has emphasized a point with which I am much in sympathy, namely, that if a level lesion is found with subarachnoid obstruction exploration should be performed. A level lesion with subarachnoid obstruction should always be explored.

As to Dr. Penfield's comment concerning the source of the xanthochromia and increased protein, I meant to imply that the source of both was from the congested veins of the spinal cord, and that it occurs whether the tumor is extradural, a chondroma, or a cold abscess; or the source can be from any condition that obstructs the circulation of the veins of the spinal cord. It is generally accepted that obstruction of these veins gives rise to increased protein and xanthochromia.

Concerning Dr. Rosenheck's question why, after so-called meningitis of childhood of the meningococcus type, adhesive arachnoiditis does not occur, I have no explanation. It is not known that various organisms give rise to various types of tissue reaction; witness the type of membrane formed by the diphtheria bacillus, and certain types of streptococcus. It is known that tissue reactions differ in various processes. I think Dr. Rosenheck should ask that question of Dr. Globus and not of me. I would be glad if Dr. Globus would tell me, for I have no explanation to offer.

I do not wish any one to think that this good result is what is usually obtained. I have operated on some patients without any improvement. In the extensive processes, one can do little or nothing. Unless the adhesions can be freed, good is not likely to come of the operation whether or not it is an extensive process or a limited one. Exploration should be done when the diagnosis is established.

Spinal Cord Compression in Hodgkin's Disease. Report of a Case of Hodgkin's Disease of Thirteen Years' Duration. Dr. George A. Blakeslee.

Compression of the spinal cord from disease of the peridural tissues such as has been known to occur in the lymphoplasias of the peridural lymphoid tissue in Hodgkin's disease is rare. A few cases are described in the literature in which paraplegia was associated with Hodgkin's disease; in some a clinical report was made, without a postmortem examination, and in others with corroborative postmortem observations. In the following case of Hodgkin's disease, of about thirteen years' duration, a complicating paraplegia developed after ten years.

When the patient was 14 years of age, enlargement of the lymph nodes was first noticed in the right supraclavicular region; at biopsy it was diagnosed as Hodgkin's disease. Following roentgen-ray therapy, the cervical lymph nodes disappeared. A remission of eight years followed, during wihch the patient was in excellent health. Then a constant, dull, aching pain in the abdomen started; examination revealed a large mass in the epigastric region and a few lymph nodes in the neck. A second biopsy confirmed the diagnosis of Hodgkin's disease. Following roentgen-ray therapy, the mass in the abdomen disappeared and the symptoms were relieved.

About eight months later, sharp shooting pains appeared, radiating from the scapulae to the anterior thoracic region. Six weeks later there was complete paraplegia with sensory changes from the fifth thoracic dermatome downward, and bilateral pyramidal tract signs in the lower extremities. Following roentgen-ray treatments, the patient was able to take a few steps in about three months, and in nine months he walked with the assistance of a cane.

Two years later, he entered the Post-Graduate Hospital with fever, increased difficulty in walking, weakness, loss of weight, and large glandular masses in the left and right axillary and pectoral regions. Histologic examination of a gland from the right axillary region showed the characteristic tissue of Hodgkin's disease. Neurologic examination revealed spasticity in gait, bilateral pyramidal tract signs and sense impairment of the posterior column in the lower extremities. The axillary, pectoral and cervical regions of the body were tested by roentgen-ray exposure, and the glands disappeared. The patient was advised to live in the country, and gained 20 pounds (9 Kg.); his strength increased and locomotion improved.

The difficulty in locomotion at the time of entrance to the Post-Graduate Hospital might have been caused by the general weakness resulting from the general adenopathy, but the presence of localizing spinal cord signs speaks for local involvement of the spinal cord.

The patient showed definite signs of compression of the spinal cord which receded under roentgen-ray therapy along with the general signs of Hodgkin's disease. Reports of necropsies prove that the lymphogranuloma of Hodgkin's disease may occur in the peridural space as a localized process. It seems fair to assume that this case of localized compression of the spinal cord in Hodgkin's disease can be explained in this way.

DISCUSSION

Dr. Ward H. Cook (by invitation): Cases of Hodgkin's disease exhibit typically symmetrical enlargement of the cervical nodes, with dyspnea and moderate emaciation. The appearance of the discrete nodes is one of the characteristic features of the glandular enlargement in this disease. The nodes may become fused by fibrosis due to secondary infection or as the result of roentgen-ray therapy. The latter fact is of interest in connection with Dr. Blakeslee's case because it has been observed at necropsy in the retroperitoneal nodes in cases in which roentgen-ray treatment was employed.

Sections of the tissue removed in 1923 present a fairly characteristic appearance of Hodgkin's disease. The deeply stained nuclei visible in the diseased tissue are those of the lymphocytes of the invaded lymph nodes. The paler cells are for the most part mononuclear cells, variously interpreted as of endothelial or reticular origin. A few of the giant cells so often described as characteristic of this disease appear scattered through the tissue. The high power lens brings out the characteristic lobulated nuclear cells more clearly. Also there are lymphoblasts and mononuclear cells of large size associated with the reticular hyperplasia.

The last biopsy specimen, taken in the spring of 1926, shows essentially the same features.

With regard to the nature of the pathologic changes, they present a typical histology, easily recognizable; yet the etiology of the process remains obscure. At the present time pathologists question whether this is an infectious process deserving the name of lymphogranulomatosis, or whether it is essentially a neoplasm. With regard to involvement of the nervous system, occasional cases have been described in the literature of such extension, chiefly from the retroperitoneal masses. A case was described recently by the late Eugen Fraenkel of Germany, in which the patient came to operation with the diagnosis of a tumor of the spinal cord. The ordinary signs of Hodgkin's disease were not observed, because the glandular involvement was retroperitoneal rather than cervical. Fraenkel reviews the involvement of bone in this disease and

observes that the vertebrae as well as the long bones are occasionally invaded. Involvement of the extradural tissues occurs by direct extension, either through the vertebrae or around the vertebrae. Following roentgen-ray treatment the condition is one of fibrosis, with disappearance of the hyperplastic cells. Recurrence in the same location is variable; recurrences following irradiation are more apt to be in some location other than the one in which postirradiation fibrosis has taken place.

Dr. J. H. Globus: Cases of Hodgkin's disease resulting in changes in the spinal cord are rare, and a clinical course of gradual improvement with a return almost to the normal is extremely uncommon. In two cases I have studied at autopsy there was no evidence of increased lymphoid tissue in the epidural space. I recall a case in which the entire posterior column was involved, presenting the clinical picture of tabes dorsalis. Of course, at no time was there a question of operative intervention, as there was no evidence of local pressure; it was recognized clinically as a systemic disease, a degenerative process, secondary to a constitutional disturbance. I am familiar with the case reported by Fraenkel, and there are other instances of the same character with actual involvement or increase in the epidural lymphoid tissue simulating the formation of tumors. The tumors usually occur in a multiplicity of sites, and for that reason present disseminated signs. They therefore do not present the sensory level shown in the case presented tonight. This is another reason for considering the case as unique.

Dr. Riley: I should like to ask whether this patient should have been operated on rather than treated by the roentgen ray. Why was the decision reached not to remove the tissue surgically, rather than to try the slower and possibly not so complete means of roentgen-ray therapy?

Dr. Abrahamson: I recently saw a case of Hodgkin's disease, verified by microscopic examination of a gland. The patient later developed jaundice, abdominal pains, headaches, and then progressive hemiplegia with aphasia. All agreed that a Hodgkin's deposit within the brain was being dealt with. An autopsy was refused.

Dr. Blakeslee: In answer to Dr. Riley, the treatment of Hodgkin's disease has been going through certain phases for many years. Several years ago vaccine treatment was tried; then arsphenamine was tried, and this has been followed by roentgen-ray and radium therapy. Surgery seems to have been practiced less frequently in recent years. The decision to use roentgen-ray treatment in this case, I think was a good one, because of the excellent results in the treatment of the adenopathy prior to the onset of the compression myelitis.

Personality Make-Up of Epileptics. Dr. J. Notkin (by invitation).

In 1919, in a paper presented at the Convention of the American Psychopathological Association, Dr. L. Pierce Clark put the question: "Is Essential Epilepsy a Life Reaction Disorder?" and answered it in the affirmative. He went into the details of the personality make-up of epileptic persons and tried to show that it is a specific one. He enumerated the well known traits of egocentricity and supersensitiveness and noted the emotional poverty and the stiffness of mentation. He further asserted that the character faults of epileptic persons are present even before the manifestation of the seizures; he spoke of increasing slowness and diminished capacity for any kind of activity long before the epileptic disorder manifested itself. In recent years he formulated the conception that epilepsy should be looked at more from a psychobiologic

aspect, and regarded it as an outflow from a homosexual component which is not sublimated or accepted. He considered the epileptic make-up as being deep rooted in narcissism, which results from poorly repressed homosexuality. He claims that the extroverted sexual life of epileptic persons ceases somewhere between 25 and 30 years of age and that the whole libido is then focussed on the ego, making larger and larger the innate narcissism.

MacCurdy, who made an extensive study of epileptic deterioration, concurs generally with the early conceptions of Clark. In Europe, Franziska Minkowska, in a paper contributed in the Festschrift on the occasion of the twenty-fifth anniversary of the Professorship of Eugene Bleuler, advocates alongside the schizoid and syntoid types of personality a third one which she called the epileptoid. However, making a study of epileptic heredity rather than of the epileptic make-up, she never went into details of the character traits and generally spoke of already full-fledged epileptic persons. Otherwise, there is little in the literature in regard to this problem except by Fischer and Kretschmer, who both causually remarked on the occurrence of the benign type of personality and the schizoid make-up in epileptic persons.

I have had an opportunity to study the make-up of nonpsychotic epileptic patients in a general hospital (New York Post-Graduate Neurological Clinic) and was surprised to find in a great majority nothing or little of the so-called epileptic make-up. I then decided to review the epileptic material at the Manhattan State Hospital and selected a group of 150 cases—seventy-five men and seventy-five women—in which a more or less reliable life history was available. Here again I was impressed with the frequent occurrence of a benign type of personality in epileptic persons. I attempted then to tabulate various factors in each case and to see if there was any correlation between them and the personality make-up. The age at the onset of the first convulsive seizure has apparently a direct bearing on the type of personality. Whenever the convulsive seizure appeared in infancy or early childhood, the make-up was decidedly epileptoid; the more remote from infancy the first manifestation of epilepsy, the more benign the make-up. Pure epileptoid traits were found in 16.6 per cent of cases in which the convulsive seizures began before 12. Even in this group a small number presented an admixture of a few benign traits and a few showed a pure syntonic personality—usually cases in which the convulsive seizures were infrequent or with intervals of many years' duration. With onset above the age of 12, and in single cases below that age, 19.3 per cent showed a normal constitution up to the first manifestation of the epileptic disorder and then a change to an epileptoid, and in 8 per cent a change to a schizoid type of personality. Then followed a small group with a syntonic type of personality with a slight admixture of epileptoid or schizoid traits (4.6 per cent). In the majority in this group the onset occurred after the age of 12. In the largest group, 37.3 per cent of those with a pure syntonic type of personality, all except three were cases with the onset of convulsive seizures after the age of 13. There was an alcoholic history in a number of these cases. It is interesting also that in about 8 per cent of pure schizoid types the epileptic manifestations appeared between the ages of 11 and 54; this was also true in 4 per cent with pure traits of psychopathic personality. One may be justified in explaining the occurrence of these various types of personality in epileptic persons by the fact that when epilepsy develops early there is little time for the epileptic to develop a personality; what then takes place is the epileptic mental reaction. This may be substantiated by the striking similarity of these traits in all cases and the more striking resemblance of this reaction to the

psychotic traits in which irritability, egocentricity and supersensitiveness have reached their limits.

In cases with a schizoid type of personality, there is a tendency for the patients to withdraw from the outside world because they are conscious of their affliction. The pure schizoid types of personality will then easily develop, with the onset of convulsive seizures at puberty and later. One must, however, think of the possibility of a primordial schizoid personality which has been awakened and brought to the surface by the epileptic reaction. It is interesting to note that the psychotic reactions are characterized by epileptic traits with an admixture of hallucinatory and delusional elements. With the onset of convulsive manifestations in later years, there is an admixture of epileptoid and schizoid traits; the former are to be considered as the result of the epileptic reaction, the latter as the awakening of the latent schizoid tendencies.

DISCUSSION

Dr. Osnato: I think this paper is extremely important. Under the leadership of Dr. Clark, there has been a definite tendency to explain idiopathic epilepsy on the basis of a life reaction, and the tendency has been extended to the point of treating these patients by psychotherapy on the theory that epileptic seizures constitute merely one portion of the general life reaction based on certain psychopathologic situations. It seems clear from Dr. Notkin's paper that the cart has been put before the horse. If Dr. Notkin's work proves anything, it seems fair to say that his conclusions are two: (1) the so-called epileptic make-up is the result of epileptic seizures, and is therefore the protective reaction of a person which results in the development of hypersensitiveness and irritability and egocentricity, and not that the epileptic seizures come because the person is possessed of these personality traits; (2) the so-called epileptoid make-up does not make itself manifest in patients who have developed seizures rather late in life; these show the rather ordinary run of personality make-up either schizoid, syntonic, or normal. It is hard to exaggerate the importance of this work. It brings us back to the more materialistic method of investigating epilepsy.

Dr. S. P. Goodhart: In your observation of 150 patients, all of whom were subject to institutional treatment and in whom I presume there was a psychosis for which they were brought to the Manhattan State Hospital, was there any common psychic factor that stood out? For example, would the patient with paranoid trends or the manic or depressed types show definite variations from the direction of the psychosis, and was there any special feature that appeared to characterize all the groups?

Dr. Notkin will recall a patient I sent to him from Bellevue, and who is now under his direction, whose reaction was typically hypomanic, though the psychotic state appeared within a short time after a mild convulsive seizure. The psychotic reaction was not that of the epileptoid equivalent, nor was it of the postepileptic state. Amnesia or confusion was not present; the distinctive flight of ideas and typical manic behavior brought her to the psychopathic ward at Bellevue Hospital. It would be of interest to know whether in psychotic episodes the epileptic personality, so-called, really has any distinguishing characteristics. I believe I share the general opinion among the majority of psychiatrists that this epileptic "personality" is a term to conjure with.

Dr. Joseph Smith: These personality studies are always interesting. The late Dr. August Hoch years ago presented a paper before the Neurological Society dealing with studies in manic-depressive conditions and dementia

praecox, and he came to the conclusion that the make-up of certain persons tends toward the dementia praecox type of others, toward the manic-depressive type of reaction. It was absorbing, but I do not believe that later observations have tended to substantiate that view or that in all cases it is typically so. The traits here observed should be regarded as symptoms of the disease, which does not manifest itself clearly until years later. A manic make-up is only discovered after the symptoms have become so pronounced that differentiation from dementia praecox is easy. Then one discovers in the patient's previous history certain traits that are named constitutional manic or schizoid traits. These are interesting, but they throw little light on the subject. A more fundamental question is: To what earlier "constitution" do these later traits owe their existence? In many cases of epilepsy one would consider that reactions have a great deal to do with situation in life, with environment, and educational niveau. Since these vary markedly, the personality make-up will also vary tremendously; one cannot, in an off-hand manner, divide the epilepsies into two great groups, the manic and schizoid. The characteristics of individuals go far beyond any limits set up by classifications.

Dr. Philip R. Lehrman: Did Dr. Notkin make individual studies of these patients? How did he arrive at the conclusions? Were they made by mass studies, or did he make personal studies after the manner of Dr. Clark?

Dr. Charles Bernstein: About two or three months ago, Dr. Duren, of the Syracuse State School, reviewed the subject of epilepsy. I have been interested in this attempt to attach to the epileptic patient an epileptic personality. Whenever epileptic patients are handled early, trained individually in an interest in life (and not allowed to deteriorate as so many do when held in large numbers in institutions), given vocational training and an opportunity for natural human activity, this so-called epileptic personality, irritability and excessive sensitiveness do not develop. If they have the opportunity to be active along normal lines, epileptic children do not show these personality traits.

Dr. Notkin: In answer to Dr. Goodhart's question, in the cases in which the seizures were infrequent or came on later in life, there is little or none of the epileptic make-up. On the other hand, these traits are evident in cases with the onset of epilepsy in infancy. I know the patient Dr. Goodhart referred to; she shows a few traits of the epileptic make-up. She is always insisting on certain things, making all kinds of demands of the physician. However, I have not had much opportunity to make a detailed study of her so far.

In answer to Dr. Lehrman's question, the study was based on the life histories with observation and examination of each individual case. I did not analyze each case but tried to study the details of the adjustment in life. When the history seemed to be insufficient, the social workers of the hospital seured additional data. Relatives or friends of the patients also have been interviewed at the hospital.

Medullary and Pontile Syndromes. Dr. Rubin A. Gerber (by invitation).

This paper, essentially clinical in character, correlates the symptomatology and clinical observations in several cases of medullary and pontile lesions with the anatomicophysiologic characteristics of the brain stem.

Case 1.—This case presents the syndrome of the nucleus ambiguus and the nucleus hypoglossus (syndrome of tabia—syndrome of Horner). A man, aged 64, complained of inability to swallow and drooping of the right eyelid, which came on suddenly. Subsequently, the tongue became "thick," and

he was unable to speak or swallow. Speech returned, but swallowing of liquids was difficult with regurgitation through the nose. The important neurologic observations were limited to disturbances of the tenth and twelfth cranial nerves, the reflexes, motor and sensory examination of the extremities giving essentially negative results. The right side of the palate and of the tongue were affected. There was the additional complicating factor of right enophthalmos, myosis and ptosis.

The fundi showed some senile perimacular changes and some angiosclerosis. Cocaine dilated the left eye widely, the right much less. The left vocal cord was involved. Fluoroscopic examination showed an apparently partial obstruction in the upper esophagus, with moderate dilatation above this point. The blood pressure was 100 systolic and 78 diastolic; laboratory tests gave negative results. The patient became progressively worse and died of some pulmonary complication early in January, 1926.

Comment.—The lesion in this case was probably thrombosis, affecting the medulla. Involvement of the hypoglossal and vagus nerves explains the tongue and palate symptoms; extension of the lesion dorsad to include the pupillary center in the medulla gives the syndrome of Horner—the ipsilateral enophthalmos, myosis and sympathetic ptosis. The roentgen-ray observations are readily interpreted as the result of slight failure in the action of the musculature of the upper portion of the esophagus, innervated, as is the larynx, by the vagus. Thus, the entire symptom complex is explained. In the absence of all sensory and of other motor symptoms, the absence of peripheral lesion of the twelfth and tenth nerves at the cervical level, and the fact of the simultaneous involvement of the tenth and twelfth nerves, point to the localization in the medulla oblongata.

Case 2.—This case shows the syndrome of the nucleus ambiguus and spinal fillet complicated by the syndrome of Horner (the syndrome of Avellis). A man, aged 56, noted closing of the left eye, and on the following day difficulty in swallowing with regurgitation of fluids through the nose. The voice changed to a whisper. On March 18, he noticed that sensation caused by the wind blowing through the window of his room felt warm on the right side of the face and different from the sensation of the left; when bathing, cold water felt warm on the right side of the face, and on the right upper and lower extremities. A few days later, fluids ceased to regurgitate through the nose and he swallowed with less difficulty. The essential symptoms were limited to the status of the sensory and cranial nerves. These showed the classic loss of pain and temperature sense in the right arm and diminution of pain and temperature sense over the right trigeminal area. The fundi showed some angiosclerosis. There was the additional complicating Horner's syndrome on the left. The uvula pointed to the right. There was paresis of the left side of the palate, with impairment of the left pharyngeal reflex. Laryngoscopic examination showed almost total immobility of the left side of the larynx, with impairment of swallowing. There was an increased secretion of tears from the left eye. Perspiration following a hot bath showed profusely on the right side of the face and little on the left. A 4 per cent solution of cocaine produced marked dilation of the right pupil, with exophthalmos and widening of the palpebral fissure, but without any change in the left eye. The heart rate ranged between 80 and 108. The blood pressure was 180 systolic; 100 diastolic. Laboratory tests were all normal.

Comment.—The lesion was probably hemorrhage in the radicular branches supplying the medulla oblongata. This is borne out by the simultaneous involve-

ment of the supply of the nucleus ambiguus to the larynx and palate, and of the spinothalamic tract (spinal fillet), which lie close together in the lateral white column and are susceptible of damage by a small lesion. This caused the loss of pain and temperature sensibility in the entire opposite half of the body, including the skin over the scalp to the interauricular line. This constitutes the syndrome of Avellis, in this case complicated by the syndrome of Horner, produced by a slight dorsal extension of the hemorrhage to include the pupillary center.

CASE 3.—This case showed the syndrome of the nucleus ambiguus, pyramid and spinal fillet. A man, aged 48, complained of difficulty in swallowing, hoarseness, pain in the right side of the larynx, pain in the right pectoral region and emaciation. The first symptom noted was pain in the right side of the larynx six months previously. The onset was gradual. The patient believed he was constantly getting better. Additional data showed that there was no difficulty in swallowing and that the pain in the right pectoral region was present at the same time as the pain in the right side of the larynx; gradually, also, hoarseness of voice and some difficulty in talking were noted.

Examination.—The voluntary motor system showed definite weakness of the right arm and leg; the right reflexes were greater than the left; transient clonus was obtained on the right and left, the right being the more enduring. Sensory examination did not reveal any objective changes, except that the pain sense was slightly less on the left than on the right; temperature sense was normal; other modalities were normal. The pupils reacted to light and in accommodation promptly. There was an old left external strabismus. The uvula pointed to the left. There were no marked differences in the motion of the palate, though there was greater elevation on the left than on the right. Also the muscles going from the palate to the fauces on the left showed greater contractile power than those on the right. All other cranial nerves were normal. The fundi were normal. The systolic blood pressure was 138; diastolic, 96. Subsequent examinations disclosed practically the same condition in the palate and vocal cord, and in the motor and sensory examination. At a later date, temperature stimulation seemed to be better interpreted on the right than on the left, but distinct loss was not present. Roentgen-ray and laboratory tests done at other institutions were all said to give negative results. Laryngeal examination showed a paralysis of the left vocal cord.

Comment.—This case does not clearly indicate the etiologic factor responsible. There is some likelihood of thrombosis, as there was no marked disturbance in consciousness and the onset seemed to be gradual. Apparently, the involvement of the spinal fillet is not grave; the symptoms, though distinct, were not the typical complete loss of pain and temperature senses that follow lesions of the spinal fillet. The disturbance of the palate and vocal cord was permanent and decisive. Affection of the nucleus ambiguus and the spinothalamic path easily occur conjointly. A complicating factor in this case is the involvement of the pyramidal tract. The lesion is probably in one of the radicular arteries supplying the medulla, which may easily cause the picture described as regards the syndrome of Avellis—characterized by laryngoplegia and palatoplegia associated with contralateral pain and temperature disturbance. Perhaps an additional lesion occurred to account for the disturbances in the pyramidal tract.

CASE 4.—This case showed the syndrome of the vestibular nucleus. A man, aged 49, complained of attacks of vertigo. He also complained of "noises in the

right ear" during the attacks. He had had a similar attack about five years previously. Before the first attack, he had had pneumonia which left, as a sequel, cardiac disease. He subsequently suffered from attacks of vertigo, on and off, for two years. The general neurologic status was completely normal, with the exception of vertigo when he was about to lie down. A few nystagmoid jerks were noted. The disks were very pale. Bone conduction was greater than air conduction equally on the right and left. There was no middle ear complication. The systolic blood pressure was 110; diastolic, 60. The pulse rate was 82, with occasional intermission, the irregularity occurring about every six to ten beats. A relative described the acute paroxysms minutely, stating that the patient became pale with moist, clammy perspiration on his forehead; he complained of being dizzy, with the bed turning round and round. Occasionally he would vomit. It would take three or four days before he felt well enough to resume his usual work.

Comment.—From the acute paroxysmal nature of the disease, one would infer a transitory lesion, especially as there is good health between the attacks. The etiologic factor is probably an angiospasm in the region of the vestibular nucleus in the medulla. Since the sense of hearing is implicated, one would think of Menière's disease. There is, however, no evidence of disease of the middle or internal ear, though the symptoms are exactly similar in the latter disease. Other special or neurologic symptoms were not present, except lost equilibratory control. This fact, in the absence of involvement of the internal ear, argues for the diagnosis of medullary involvement, especially when the cardiac condition is taken into consideration.

Case 5.—This case showed abducens alternate hemiplegia. Pontile syndromes are particularly characterized by the alternate character of the lesion. A child, aged 4 years, presented a paralysis of the left external rectus muscle which came on one week before, after an attack of measles. This was apparently sudden in onset and no change had been noticed since the onset. Other illnesses or abnormalities were not present.

Examination showed the right reflexes uniformly increased over the left and Babinski and Chaddock signs on the right. Sensation was normal. Of the cranial nerves, only the paralysis of the left external rectus was noted. The fundi were normal. Dr. Reder's report of the condition in the eye confirmed the left external rectus palsy. The diplopia increased as the patient looked to the left; tested with light, the images in a horizontal direction became wider and wider apart. There was no error in refraction. The diagnosis was: nonrefractive convergent squint due to paralysis of the sixth nerve; right eye normal in every particular.

Comment.—The etiologic factor here is probably toxemia due to measles, with a thrombosis in the caudal portion of the pons. A small lesion here would involve both the emergent fibers of the sixth nerve and the pyramidal tract, causing a syndrome of alternate abducens hemiplegia.

Case 6.—This case showed hemiplegia alternans hypoglossus et facialis. A man, aged 55, complained of difficulty in walking and weakness in the right leg. He did not have any difficulty in swallowing, but slight difficulty in talking clearly.

Examination.—Definite left facial weakness, ataxic gait, with dragging of the right foot, reduced associated movement of the right arm in walking, and right hemiplegia were noticed. Reaction of degeneration was not present in the muscles of either the right or left extremity. Sensory examination gave

completely negative results. There were evidences of marked arteriosclerosis. Arcus senilis was present. The left nasolabial fold was less marked than the right, and the angle of the mouth was lower on the left than on the right. The upper branch of the facial was apparently normal. There was an indefinite diminution of movement of the palate in the right during phonation. The hypoglossal nerve showed involvement: the tongue deviated to the left; it showed some corrugations on the left, with the left half smaller than the right. The systolic blood pressure was 220; diastolic, 110. The various laboratory tests all gave negative results; Bárány tests were negative.

Comment.—The lesion was undoubtedly a hemorrhage; this is borne out by the arteriosclerotic changes in the vessels and the high blood pressure. The site of the lesion is in the medulla on the left side involving the left pyramid and the left hypoglossal nucleus. There is a further slight complication in the facial nerve of the left side; whether it is the residual of some old condition it is hard to say, as there was no reaction of degeneration. It is more likely that there was a progression of the symptoms with the lesion extending across the midline, or it may be an irritative or pressure phenomenon due to interference with the circulation in the region of the facial nucleus.

Case 7.—This case showed hemiplegia alternans facialis complicated by subsequent ipsolateral hemiplegia. It also shows the continuity of the etiologic factors in producing ever extending lesions. The woman, aged 54, complained of twitchings all over the body, general nervousness, shooting pains in the head and hot flushes all the time; the last began first five years before but had been worse of late. She complained particularly of dizziness, saying she could not turn in bed; everything would turn round her, and she would feel nauseated. When walking she would fall over, more to the right than to the left, and had to be careful. The first symptom was dizziness; this had been gradually getting worse.

Examination.—The patient held the head stiffly; she walked normally, but carefully and slowly. Coordination tests gave normal results. The reflexes on the left were more marked than on the right. The abdominal reflexes were not elicited because of obesity. There was a questionable Babinski sign on the left and none on the right; fanning was present on both sides. All confirmatory signs were absent. The Hoffman sign was present on both sides. The sensory examination gave negative results. The ocular fundi showed evidences of arteriosclerosis. The right palpebral fissure was wider than the left. There was a right lower facial paresis. The general systemic examination revealed: obesity; pulse rate, 84; systolic blood pressure 180; diastolic, 80; hot flashing over the face.

On March 26, 1926, there were signs of slight hyperreflexia on the right, greater than on the left. The Hoffman sign was more active on the right than on the left. The achilles reflex was more active on the right than on the left. The systolic blood pressure was 200; diastolic, 100. The patient complained of dizziness and said that she had to change posture slowly, as the slightest movement was accompanied by severe pains in the head. Laboratory tests gave negative results with the exception of the urine, which indicated some slight nephritic condition.

Comment.—This patient had an incomplete facial paralysis on the right with left hemiplegic signs, and subsequently, in addition, an ipsilateral hemiplegia. An organic lesion may be masked by neurotic symptoms. This patient undoubt-

edly had neurotic symptoms associated with the menopause (hot flushes, nervousness, irritability, etc.), but also grave organic symptoms. The lesion was a hemorrhage in the caudal half of the right side of the pons, involving the facial fibers and the pyramidal tract. The sixth nerve is frequently involved in lesions of this kind but apparently escaped here. Subsequently, another vascular insult occurred and caused increasing signs of involvement of the right side. There was apparently no nuclear or infranuclear involvement of the facial nerve.

DISCUSSION

DR. ABRAHAMSON: It is not fair to discuss a paper of which the author has only read fragments. I think Dr. Gerber has presented an unusual group of cases.

DR. OSNATO: It is difficult to make this material interesting or attractive. The paper is extremely well and carefully written, and will be well worth reading. In the case in which the esophagus was involved as part of the syndrome, a provisional diagnosis of carcinoma of the esophagus had been made; it was only after the patient had been studied from the neurologic standpoint that the real condition was understood.

Book Reviews

CHILD GUIDANCE, with an introduction by DR. THOMAS W. SALMON. By SMILEY BLANTON and MARGARET GRAY BLANTON. Pp. 294. Price, $2.25. New York, the Century Company, 1927.

In the present volume, the authors have made a commendable effort to fill a gap in the literature on child guidance. Their aim, as expressed in the foreword, is to treat of "that single phase in which the normal child is the problem." As the reviewer sees it, they are not interested in discussing, for example, how to attack the problem of the child of 4 who suffers from enuresis; they are primarily interested in how to attack the problem, which all parents face, of so handling a child's life that he will develop control of his urinary functions at the normal time in his growth. In other words, they have tried to write a book that can be used as a guide for keeping children on the proper path of habit and personality development. They have attempted a difficult task and have succeeded for the most part admirably.

There are three parts to the book. The first deals with the physical training of the child, and the problems of eating, sleeping, walking and talking, control of the excretory functions, and the training in sensory appreciation are discussed. These chapters are practical and concrete and contain many valuable suggestions. Sole exception may be taken to the chapter on "Learning to Eat," in which the authors make such questionable statements as that on page 38: "Often it becomes necessary to force the feeding, and in such cases it is well to hold the nose for a moment until the mouth is opened and then insert a small spoonful of food"; or again, on page 35: "We feel that it can safely be assumed that there is some fault in the metabolism of the child or some anomaly of the bone and cartilage about the mouth in those cases in which thumb-sucking (or finger-sucking) is present after meals and that the thumb-sucking may better be considered as a symptom of some such anomaly than as a bad habit." The suggestion on page 40, "Any change in attitude or intention (refeeding) on the part of the parent should be talked over with the child, and the reasons for the new rules explained," is scarcely sound if one realizes that feeding difficulties occur at an age when logical reasoning is beyond the capacity of the child.

The second part of the book concerns itself with the social side of the child's development. In it may be found interesting and worth-while discussions on how the home and its routine should be organized, how the adjustment to the group may be prepared for, how the problem of sex instruction may be handled and how the question of discipline is to be treated. In these more general and abstract situations there is an occasional tendency to generalize and to give warnings without being specific as to the steps to be taken to avoid certain difficulties. The advice to tell all about sex at the first questioning on the part of the child, even at the risk of confusing him, is a doubtful policy. It emphasizes sex as pointedly as does the usual policy of silence.

The last two chapters of part two seem somewhat apart from the others in purpose. Chapter XIV on "Nervousness" is excellent, with many actual case illustrations to drive home the fact that nervousness is so often a symptom and not a disease. Chapter XV on "Intelligence" is a sane and timely presentation of the influence of emotional factors on intellectual achievement.

Part three is an endeavor to discuss ideal character traits and what they mean. It contains many excellent ideas, but it suffers from the fact that there is too much talk of the goals to be attained and too little of the steps by which these are to be reached. It will inflict further burdens on the conscientious parents without giving adequate assistance. The personality study at the end seems somewhat extraneous and could be dropped without detriment to the book.

In the main, the book is entirely safe and can be recommended to parents, especially those whose children are of the preschool age. It is not a volume to be used for the more advanced workers in the field, but is more an effort to take the material already known and to organize it in acceptable form for the practical use of parents, physicians, and other people who must deal with children, but cannot devote their entire time and energy to the study of the task. For them, it should have a considerable value.

ANATOMIE DES NERFS CRANIENS ET RACHIDIENS ET DU SYSTÈME GRAND SYMPATHIQUE, CHEZ L'HOMME. By A. HOVELAQUE. Two volumes, with 873 pages and 89 figures and 121 plates. Paris: Gaston Doin et Cie, 1927.

Hovelaque has assembled in these two volumes an enormous amount of information about the peripheral nervous system. It is largely descriptive, minutely detailed, and up to the minute as regards recent advances in anatomic science. A sixty-five page bibliography attests the prodigious library research of the author. The work is well printed on a fair quality of paper and is satisfactorily bound. The illustrations throughout give an impression of excellence because of the modern technic of the artist, but careful examination and comparison with other anatomic illustrations and with the relations seen in dissections reveal a carelessness that is sometimes astounding. Thus in one instance (plate 5) the optic chiasm and the oculomotor nerves are shown with the base of the skull, but no sella turcica appears in the picture. Moreover, convex contours appear concave. Elsewhere, in the effort to bring out salient details, undue liberties have been taken with the relative size of surrounding structures. Since many illustrations admittedly are taken from other authors, the general effect of such garbled versions is unfavorable. It would be well if the author had chosen his illustrations with greater care.

Few will find fault with the accuracy of the text. Not only the usual relations, but also the variations, are discussed in detail. It is interesting to note how much is owed to older anatomists like Henle and Cruveilhier. In certain sections the latter's name is found on almost every page. A reference to Cruveilhier's work, however, shows how necessary this is, for even if his illustrations are wood-cuts, the text, published more than fifty years ago, still remains a monument to his industry and powers of observation. As the philosopher has well expressed it, we see farther in these days not because we have better vision but because we stand on the shoulders of our forebears. Respectful references to these older anatomists are obligatory. From the dates encountered time after time in the bibliography, a first impression is gained that the search after nerves ceased some time before the beginning of the present century. Probably the foundations were well laid by that time, but a host of younger anatomists has arisen who do not always take the word of the ancients for law, and who seek for themselves. It is these men who have been instrumental in untangling the perplexing relationships of the lower cranial nerves, and Hovelaque has done science a service in collecting their observations and comparing the results of different investigators, so that the reader

may have a clearer idea of the approximate truth. The author refers occasionally to some careful work that he has done along certain lines, but he does not force his views against those of others, and throughout retains a fairly judicial attitude.

As a reference work for minute details (discounting the illustrations) the volumes are of great value. As a stimulant to the imagination they are nil. Cruveilhier's work is more interesting. The author has collected a mass of facts as in a catalogue and has presented them coldly and logically, but almost without a trace of imagination. The meaning of the relationships, the reason for the apparently extraordinary course of certain nerves does not lead him an inch from the course of straight descriptive anatomy. The functional relationships of nerves, their course of development and their phylogenetic aspects receive little or no notice. The method of action, the nature of the nerve impulse, the fact and explanation of overlap of spinal roots, and the philosophy of metamerization—all questions that aid in the understanding of the nervous system and its *raison d'etre* and lighten the task of the reader—are neglected. It is admitted that such topics, if carefully treated, might swell the volumes beyond the bounds set by the publishers, yet one would think that 873 pages would admit a little liberty in the choice of such subjects.

Taking the work as it is, the greatest fault is with the illustrations, and the greatest good, the bibliography. The text is clear and concise as in all French works, and the volumes are mines of information for those interested in the purely morphologic characteristics of the peripheral nervous system. An unusually good index greatly facilitates reference to particular sections. A little greater clarity would result from the use of the Basle nomina anatomica terminology throughout.

L'EXCITABILITÉ EN FONCTION DU TEMPS. LA CHRONAXIE, SA SIGNIFICATION ET SA MÉSURE. By LOUIS LAPICQUE. Pp. 371. Price, 45 francs (about $2.50). Paris: Les Presses Universitaires de France, 49 Boulevard St. Michel, 1926.

For many years neurologists have needed more precise methods for investigating the series of physiologic and pathologic changes that follow injury of the motor nerves. The familiar reaction of degeneration, as studied by means of faradic and galvanic currents, has proved rough and uncertain, and is all too often of little service in arriving at a satisfactory prognosis. Studies on nerve regeneration by means of accurate determinations of the time constants of excitation have, however, especially in the hands of Bourguignon (Chronaxie chez l'homme, Paris: Masson & Cie, 1923), proved enormously fruitful both as an aid in diagnosis and in the establishment of accurate prognosis. For the elucidation of the time factors of the excitatory process of nerve and muscle, the scientific world is indebted largely to the exhaustive investigations of Lapicque and his collaborators in the physiology laboratory at the Sorbonne. In the course of twenty-five years of unremitting labor in this field of work, Lapicque has provided the mathematical and physiologic basis on which largely rests the knowledge of the normal excitatory processes and of the pathologic variations therefrom.

The book under review sums up with admirable brevity all of the author's previous investigations in this field; in addition, he brings together and discusses critically other observations on the physiology of excitation. The monograph is indispensable to anyone who would study scientifically the physiologic and clinical aspects of nerve conduction and nerve injury. The book is written throughout with that order, clarity, and finesse so often associated with the

French mind, and it is hardly an exaggeration to say that there is not an obscure sentence in the entire work.

In his preface Professor Lapicque makes apology for the occasional use of mathematics in a physiologic treatise, but in justification he points out that since physiology, as Bernard first emphasized, is in reality the chemistry and physics of life, one cannot reason adequately in this field without a little algebra — the language of physics. And so, in simple mathematical terms, Lapicque proceeds to elucidate the physical principles of the excitatory process.

There is a brief introduction which deals with the general features of excitability. The text of the work is divided into four parts containing in all fifteen chapters. The first part is devoted to the historical aspects of the subject, beginning with Swammerdam and Galvani and carrying the subject down to Engelmann, Waller and Weiss. One reads of the law of excitation of Du Bois-Raymond — now known to be false — which held that excitation resulted from change of current intensity rather than from an effective current duration; and again one's eye is struck by a chapter (III) headed "Thirty Years of Dogmatism" (1870-1900) during which Du Bois-Raymond's law was upheld by König and Hermann without critical examination of the evidence. In this admirable historical setting, the recent developments of the subject find logical expression.

Part two is purely objective, being concerned with the requisite quantitative conditions in experiments on excitability: the relation of the intensity of a constant current stimulus to its minimal effective duration (chart V), the duration of current as determined by means of condensers and the difficulties involved (chart VI), and finally the relative ineffectiveness of currents of slowly changing intensity (chart VII). On these seemingly highly technical phases of the subject the author, in part three, bases his discussion of the theories of excitation. This section of Lapicque's monograph contains the most valuable critique on the excitatory process of irritable tissues to be found in any language. A. V. Hill's mathematical treatment of excitation is carefully analyzed and found wanting in that his equations lead to "insolubles" and "unmanageables," and, when simplified, to values quite different from those observed experimentally. Lapicque offers an alternative theory which is simpler mathematically and, though it too is beset by difficulties, is probably the most satisfactory working hypothesis at present recognized (chart VIII).

It is important in studying irritable tissues, as Lapicque points out (part 4, chart XI), to have some adequate basis for comparison of their excitable properties, and one must also be able to contrast the same tissue in different physiologic and pathologic states. In a crude way this can be accomplished by comparison of the galvanic and faradic thresholds, but occasionally relatively sluggish tissues respond to lower intensities of galvanic currents than will serve for rapidly responding tissues. However, when the duration of the exciting stimulus (galvanic) is used, these discrepancies do not occur, for the optimal stimulus of a sluggish tissue is invariably of greater duration than that of a "rapid" tissue; similarly, a muscle whose nerve is degenerating acquires an optimal stimulus of progressively increasing duration. For routine study, it is necessary to use a current intensity considerably above the threshold in order to guard against adventitious alterations in tissue resistance, and one ordinarily employs a current of double the threshold intensity, the minimal effective duration thereof being designated by Lapicque as the "chronaxie."

The various aspects of this time constant are discussed at length in part 4. The author deals critically with the debated question of the influence of size

and nature of electrodes on the magnitude of the chronaxie, stating that electrode size is much less important than some authors aver. In discussing the nature of junctional transmission, he points out (with some emphasis) the fallacies in Lucas' evidence for the existence of receptive (β) substances intermediate between nerve and muscle. He then elaborates at length his own theory of "junctional" transmission, which rests on the experimental fact that motor nerves have the same chronaxie as the muscle fibers that they innervate (isochronism). The action current of the nerve, since its duration varies with the chronaxie, is held to be the adequate stimulus of the muscle fiber, and the hypothesis of receptive substances is therefore wholly unnecessary. Curare produces its characteristic effects by prolonging the chronaxie of the muscle to such an extent that stimulation of the muscle by the nerve action current becomes impossible (heterochronism). The evidence for this conception is dealt with at length, and this is by far the most convincing exposition of the theory which has yet come from Lapicque's pen.

Finally, several chapters are devoted to the technic of chronaxie determination, an account that will be welcomed by both physiologists and clinicians in all parts of the world.

An attractive feature of the book is the free use that the author has made of quotations from original sources. Those from the classical work of Nernst will be greatly appreciated, for though many have referred to his theory, few have been able to see his original work. Professor Lapicque promises to deal more completely in the future with the application of chronaxial conceptions to the problems of the physiology of the central nervous system and to cardiac physiology, both of which have a highly important bearing on pharmacology and clinical medicine. For the present one must be satisfied with guarded statements on these questions made in the final chapter.

There are a number of trifling errors in the mathematical formulas, which are usually obvious; that on page 143 should be corrected: the value $\frac{\sqrt{n}\ \sqrt{D}}{as}$ should read $\frac{as}{\sqrt{n}\ \sqrt{D}}$ in both formulas at the bottom of the page. It is regrettable that so excellent a work should not be provided with an alphabetical index. Publishers of French scientific works continue to labor under the delusion that a table of contents at the end of a book takes the place of an index.

PRACTICAL CLINICAL PSYCHIATRY FOR STUDENTS AND PRACTITIONERS. By EDWARD A. STRECKER, A.M., M.D., Medical Director, Pennsylvania Hospital, Department for Mental and Nervous Diseases, and FRANKLIN G. EBAUGH, A.B., M.D., Professor of Psychiatry, University of Colorado. Pp. 375. Price, $4.00. Philadelphia: P. Blakiston's Son & Co., 1925.

This volume of 375 pages should prove particularly useful to medical students and also to general practitioners who desire a brief authoritative description of the various types of mental disorder and the principles underlying modern methods of treatment. The authors have successfully followed the case method in presenting the subject, believing that the next best thing to actual bedside teaching is "to bring the story of the patient and his psychosis to the student through the medium of the printed page."

Chapter 1 is devoted to a brief discussion of the general principles of etiology, diagnosis, prognosis and treatment of mental disorders, with some statistics on the relative frequency of the various psychotic types among admissions to institutions. The legal requirements for the admission of patients to mental hospitals are set forth for the benefit of those who may be called on to act as examining or committing physicians.

Chapter 2 outlines the classification of mental disorders adopted by the American Psychiatric Association and now generally used by mental hospitals throughout the country.

Chapter 3 discusses methods of examination, history taking, physical examination and the mental status. The importance of the study of personality is referred to, but one misses any outline or plan of examination that the physician could follow in making such studies. In discussing the subject of summaries of cases and diagnostic conclusions for staff conferences, the authors advise that each physician working up a case should undertake a "formulation" to include the examiner's personal opinion of the genesis, precipitating factors and determining mechanisms — "the wisdom of exercising independent thought cannot be too strongly emphasized. Original conceptions, even though they may be faulty, are valuable. This frequently stimulates new lines of thought and opens up untravelled avenues of investigation."

The remaining chapters, 4 to 12, are devoted to a discussion of the various psychotic groups. A short statement of the main clinical features of each group is first given and then follows a series of well chosen illustrative cases with a short discussion of each from the standpoints of determining causes, symptoms and treatment. The neuropathologic observations in the organic psychoses are given by Dr. Winkelman.

Throughout the book emphasis is placed on the problems of internal medicine in relation to psychiatry, and the histories of the cases indicate that the patients were particularly well studied from the general physical and laboratory points of view. In dealing with the psychotic and behavioristic manifestations, the authors remain close to "objective symptoms" and state frankly that the book "does not pretend to discuss subjective psychiatry or to interpret psychotic mechanisms. . . . It is our feeling that the student must first understand objective psychiatry before he can appreciate analytic interpretation." It is not believed that the authors intend to convey the impression that the two methods of psychiatric approach stand in any opposition to each other, when at the present time it is realized more and more that all mental symptoms have a significance, the interpretation of which is essential for the understanding of any form of abnormal behavior.

In discussing the neuroses and psychoneuroses, the authors have wisely emphasized the need of considering the physical status and general medical problems presented by the patients; but, on the other hand, they take what would be considered by many psychiatrists as an ultra-conservative attitude toward the psychologic approach in the study of these disorders. They point out the danger of bringing unconscious material to the surface in every neurotic patient and think that often simple forms of psychotherapy meet the requirements. The book closes with a glossary and an unusually complete index.

As an introduction to the study of descriptive psychiatry and the practical problems of treatment and general management of mental patients, this manual forms a valuable addition to the works now available for the student and general practitioner.

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